“A PROSPECTIVE, RANDOMIZED STUDY COMPARING ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK USING BUPIVACAINE ALONE AND COMBINATION OF BUPIVACAINE AND BUPRENORPHINE IN PATIENTS UNDERGOING ELECTIVE UPPER LIMB ORTHOPAEDIC SURGERIES”

Dissertation submitted to

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY

In partial fulfillment for the award of the degree of

DOCTOR OF MEDICINE

IN

ANAESTHESIOLOGY

BRANCH X

INSTITUTE OF ANAESTHESIOLOGY AND CRITICAL CARE

MADRAS MEDICAL COLLEGE

CHENNAI, TAMILNADU

APRIL 2016

CERTIFICATE

This is to certify that the dissertation entitled, **"A PROSPECTIVE, RANDOMIZED STUDY COMPARING THE ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK USING BUPIVACAINE ALONE AND THE COMBINATION OF BUPIVACAINE AND BUPRENORPHINE IN PATIENTS UNDERGOING ELECTIVE UPPER LIMB ORTHOPAEDIC SURGERIES"** submitted by **Dr. G. ARCHANA**, in partial fulfillment for the award of the degree of Doctor of Medicine in Anaesthesiology by the Tamil Nadu Dr.M.G.R.Medical University,Chennai, is a bonafide record of the work done by her in the INSTITUTE OF ANAESTHESIOLOGY AND CRITICAL CARE, Madras Medical College and government hospital, during the academic year 2013-2016.

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DECLARATION

I hereby, solemnly declare that this dissertation entitled, **"A PROSPECTIVE, RANDOMIZED STUDY COMPARING THE ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK USING BUPIVACAINE ALONE AND THE COMBINATION OF BUPIVACAINE AND BUPRENORPHINE IN PATIENTS UNDERGOING ELECTIVE UPPER LIMB ORTHOPAEDIC SURGERIES"** is a bonafide record of the work done by me in the Institute of Anaesthesiology and Critical Care, Madras Medical College,Chennai. During the period 2013-2016 under the guidance of Prof. DR. B.KALA, M.D.,D.A.,Professor and Director of anaesthesiology and critical Care, Madras medical College, Chennai- 3 and submitted to The Tamil Nadu Dr. M.G.R. Medical University,guindy,Chennai-32, in partial fulfillment for the requirements for the award of the degree of M.D.Anaesthesiology (Branch X),examinations to be held on April 2016.

I have not submitted this dissertation previously to any university for the award of degree or diploma.

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Date: Dr.G.ARCHANA

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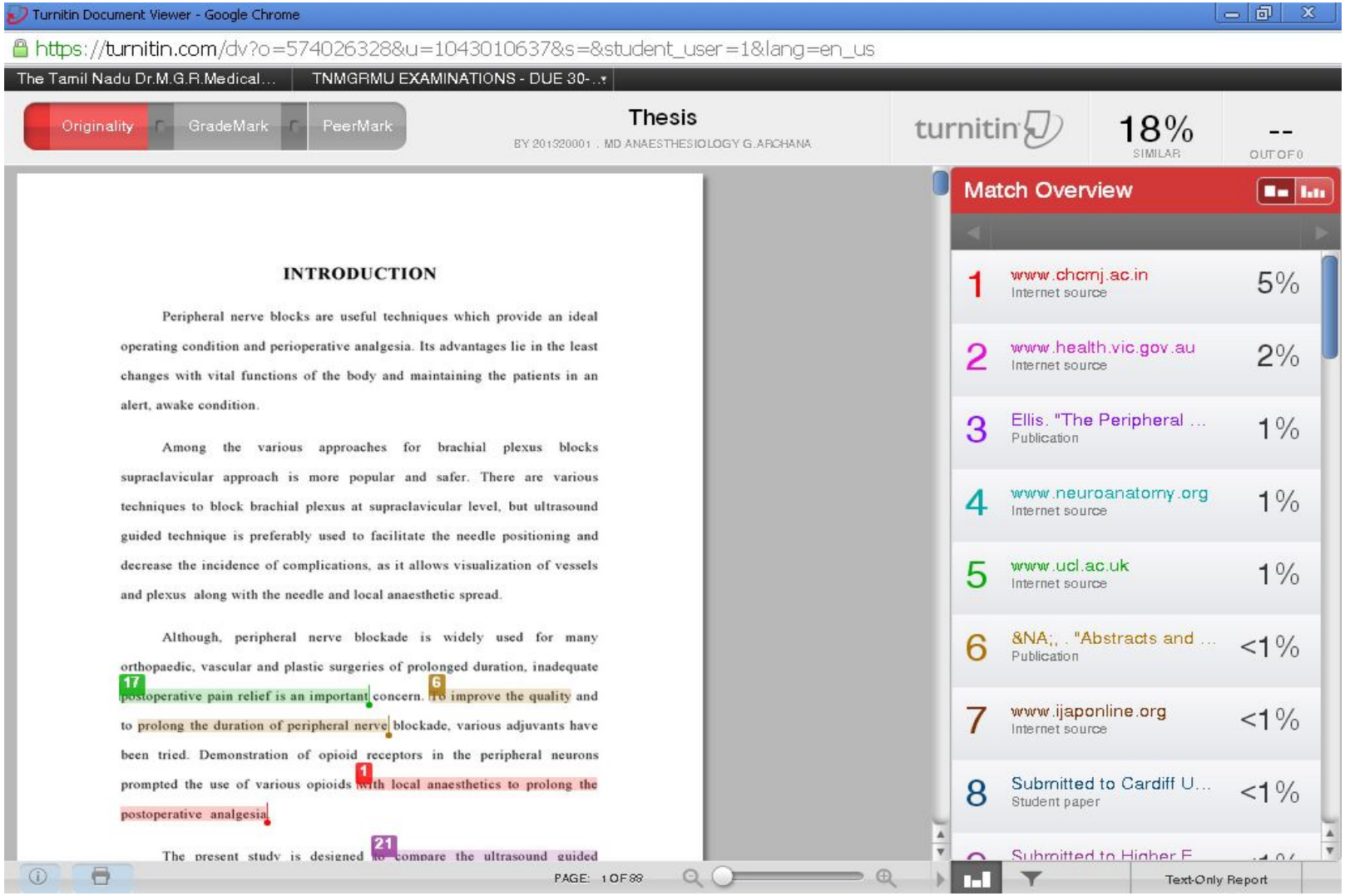
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INTRODUCTION
Peripheral nerve blocks are useful techniques which provide an ideal operating condition and perioperative analgesia. Its advantages lie in the least changes with vital functions of the body and maintaining the patients in an alert, awake condition.
Among the various approaches for brachial plexus blocks supraclavicular approach is more popular and safer. There are various techniques to block brachial plexus at supraclavicular level, but ultrasound guided technique is preferably used to facilitate the needle positioning and decrease the incidence of complications, as it allows visualization of vessels and plexus along with the needle and local anaesthetic spread.
Although, peripheral nerve blockade is widely used for many orthopaedic, vascular and plastic surgeries of prolonged duration, inadequate postoperative pain relief is an important concern. To improve the quality and to prolong the duration of peripheral nerve blockade, various adjuvants have been tried. Demonstration of opioid receptors in the peripheral neurons prompted the use of various opioids with local anaesthetics to prolong the postoperative analgesia.
The present study is designed to compare the ultrasound guided
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INTRODUCTION

CONTENTS

ABSTRACT

BACKGROUND:

Many adjuvants have been tried to enhance the analgesic efficacy of local anaesthetics in supraclavicular brachial plexus block. From previous studies, it was noted that opioids added to local anaesthetics produce comparable analgesia in supraclavicular brachial plexus block. Hence, in my study, I have compared a group of patients who received bupivacaine alone with the other group of patients who received the combination of bupivacaine and buprenorphine.

STUDY DESIGN:

A total of 60 patients belonging to ASA status I and II posted for elective orthopaedic upperlimb procedures were enrolled and randomly divided into two groups of 30 each.

Group I received 24 ml of 0.25% bupivacaine and 1 ml NS

Group II received 24 ml of 0.25% bupivacaine and 3µg/kg buprenorphine.

VAS score, onset of motor and sensory blockade were observed at fixed interval for 24 hours postoperatively.

SPSS 16.01 Version were used for descriptive, analytic and comparative statistics.

RESULT:

Onset of motor blockade delayed in group II.VAS score were found to be better in group II. Time to first rescue analgesia was prolonged in group II.

CONCLUSION:

The results showed that buprenorphine enhance and prolong the analgesic effect of bupivacaine when used for supraclavicular brachial plexus block in patients undergoing orthopaedic surgeries.

INTRODUCTION

Peripheral nerve blocks are useful techniques which provide an ideal operating condition and perioperative analgesia. Its advantages lie in the least changes with vital functions of the body and maintaining the patients in an alert, awake condition.

Among the various approaches for brachial plexus blocks supraclavicular approach is more popular and safer. There are various techniques to block brachial plexus at supraclavicular level, but ultrasound guided technique is preferably used to facilitate the needle positioning and decrease the incidence of complications, as it allows visualization of vessels and plexus along with the needle and local anaesthetic spread.

Although, peripheral nerve blockade is widely used for many orthopaedic, vascular and plastic surgeries of prolonged duration, inadequate postoperative pain relief is an important concern. To improve the quality and to prolong the duration of peripheral nerve blockade, various adjuvants have been tried. Demonstration of opioid receptors in the peripheral neurons prompted the use of various opioids with local anaesthetics to prolong the postoperative analgesia.

The present study is designed to compare the ultrasound guided supraclavicular brachial plexus block with respect to efficacy of buprenorphine in prolonging the postoperative analgesia when added to bupivacaine.

AIM OF THE STUDY

To compare ultrasound guided supraclavicular brachial plexus block using bupivacaine alone and combination of bupivacaine and buprenorphine in patients undergoing elective upper limb orthopaedic surgeries with respect to,

1) Postoperative analgesia using visual analogue pain scale.

2) Postoperative analgesic initiation time.

3) Intraoperative hemodynamics.

APPLIED PHYSIOLOGY[11],[28]

Peripheral nerves are lengthy axons of cell bodies of neurons that are situated centrally. A peripheral nerve consists of both motor and sensory fibres and are covered by 3 layers. Each nerve fibre is enclosed in a layer called neurilemma or axonal membrane. Nerve fibres are enclosed in the layer called endoneurium and these collection of bundles are covered by perineurium, outer most layer covering the perineurium is called epineurium. Based on the presence or absence for myelin sheath, it can be myelinated or unmyelinated nerve fibre.

During nerve conduction, the signal is conducted downstream the neurons by opening or closing of voltage gated ion channels and causes reversal of Resting membrane potential to action potential. The action potential formation can be divided into 5 steps.

The normal resting equilibrium potential is about -70mV and is maintained by Na+ K+ ATP ase.

1) A stimulus causes the neuron to depolarise toward the threshold potential (-55mV).

2) Once the threshold potential s is achieved all the Na+ channels open, allowing Na+ to enter the cell and the membrane become depolarised.

3) At the peak action potential (+30mV), K+ ions leak out of the cell to restore the electrical neutrality and the Na+ channels close.

4) Hyperpolarisation occurs as K+ continues to leave the cell and makes the membrane refractory to further stimulation.

5) Restoration of resting membrane potential is achieved by Na+/ K+ transporter.

ACTION OF LOCAL ANAESTHETICS[22]

Local anaesthetics consist of a lipophilic portion and a hydrophilic portion with hydrocarbon chain separating the both. Local anaesthetics prevent the transmission of nerve impulses by inhibiting the passages of sodium ions through voltage gated Na+ channels in the nerve membrane (conduction blockade).

This is brought about by,

1) Local anaesthetics block conduction by stabilization of the resting membrane potential, therefore the permeability changes cannot occur, Na+ and K+ ions movement are blocked thereby preventing the depolarization.

2) Unionised molecule of the local anaesthetic diffuses across the lipid layer of the axon. After diffusion, the quartenary amine binds with the receptor of Na+ channels when it is in inactivated state and depolarization is prevented by inhibiting the conformational change and stabilize the membrane in this inactivated resting state.

PAIN PATHWAY[11]

Nociceptors are specialized receptors that generate nerve impulses in response to noxious stimulus and brain interprets this as pain. Nociceptors are free nerve endings of primary afferent fibres of Aδ and C nerve fibres. They are distributed throughout the body and can be stimulated by chemical, mechanical and thermal stimuli. During tissue damage, inflammatory mediators like bradykinin, prostaglandins, cytokines and hydrogen ions are released and stimulate the nociceptors directly. Primary sensitization occurs which reduce the activation threshold of nociceptors.

PRIMARY AFFERENT FIBRES:

Noxious stimuli are carried by Aδ and C fibres and non noxious stimuli are carried by Aβ fibres.

Aβ – They are highly myelinated with large diameter and low activation threshold. They respond to light touch. Apart from this, they will recruit inhibitory interneurons and inhibit nociceptive input at the same spinal segment.

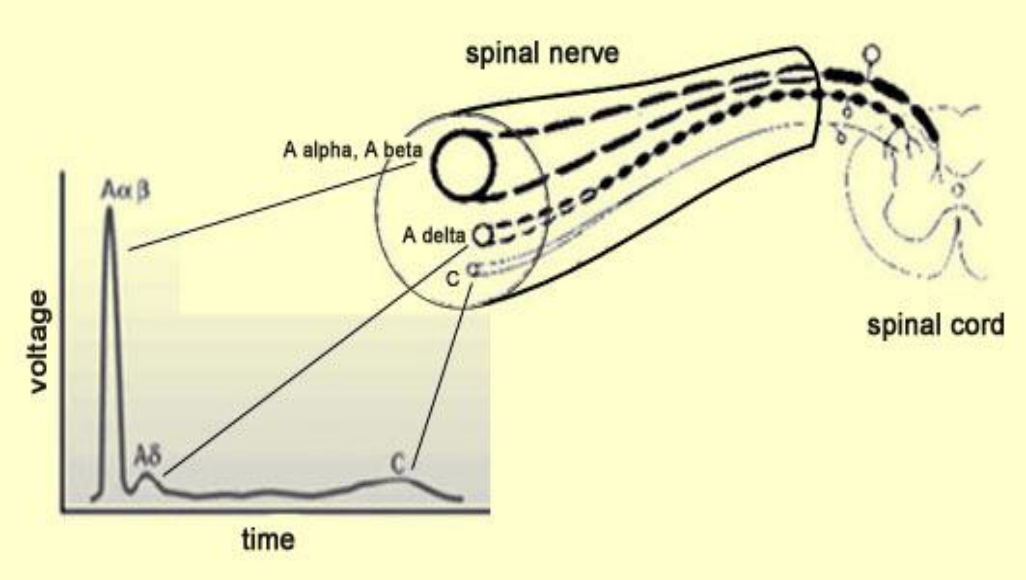
Aδ - They are lightly myelinated with smaller diameter. They conduct more slowly than Aβ fibres and carry rapid sharp pain.

C – They are unmyelinated fibres with smallest diameter and hence they have slowest conduction. They carry slow, burning pain.

Primary afferent neurons release excitatory neurotransmitters like substance P and glutamate.

Transduction - A delta fibres and C fibres

A-Delta fibres	**C- fibres**
myelinated	unmyelinated
fast (first) pain -conduct at 5-35m/sec	Slow (second) pain – conduct at 0.5-2.0m/sec
Associated with Sharp, brief, prinking pain	Associated with dull,burning, aching, prolonged pain
Well localised	More diffuse
Elicited by mechanical or thermal stimuli	Elicited mainly by chemical stimuli or persisting mechanical or thermal stimuli



SECONDARY AFFERENT NEURONS:

They are located in dorsal horns of spinal cord. Histologically, dorsal horn is divided into ten layers and are called Rexed laminae. Nociceptive

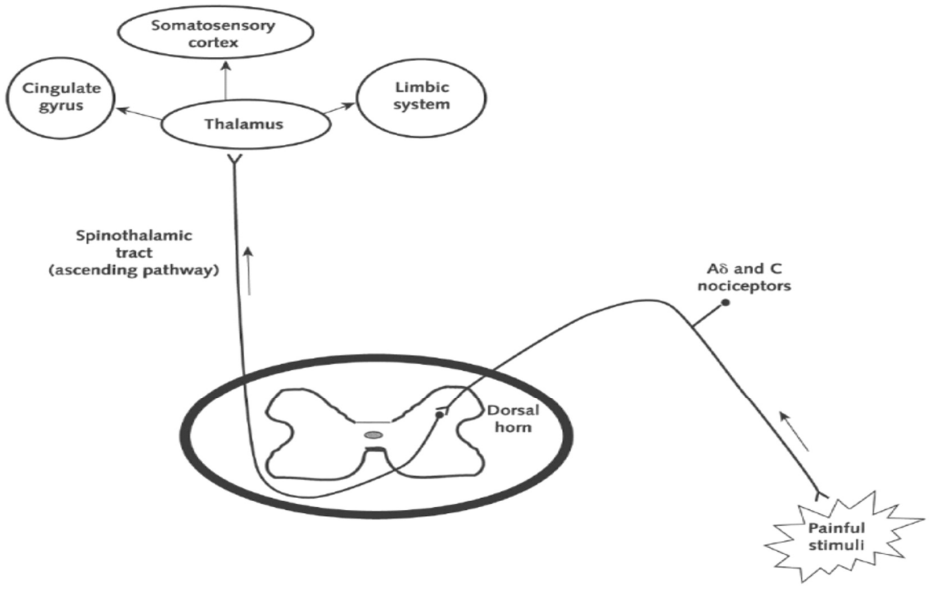
stimuli from Aδ and C primary afferent neurons transmit the impulses to secondary afferent neurons that are present in lamina I, II and V.

Secondary neurons can be divided into nociceptive specific neurons and wide dynamic range neurons.

ASCENDING TRACTS IN THE SPINAL CORD:

There are two main pathways.

1) **SPINOTHALAMIC TRACT:** Secondary afferent neurons decussate within a few segments from the level of entry into the spinal cord. Then, they ascend in contralateral spinothalamic tract and synapse in nuclei in the thalamus. From thalamus third order neurons ascend to terminate in somatosensory cortex also projects into periaqueductal grey matter.



2. SPINORETICULAR TRACT:

Secondary afferent neurons decussate and ascend contralaterally to reach the brainstem reticular formation before reaching thalamus.

ENDOGENOUS PAIN MODULATION MECHANISMS:

ENDOGENOUS EXCITATORY MECHANISM:

Spinal excitatory mechanisms:

Spinal sensitization is defined as increased excitability and spontaneous discharge from spinal nociceptive neurons to a nociceptive stimulus. This depends on the NMDA receptor activation which occurs due to sustained release of glutamate.

Descending excitatory mechanisms:

It is postulated that during nociceptive activity, there is activation of "ON" cells and inhibition of "OFF" cells. This may change the normal neuronal response to specific neurotransmitters.

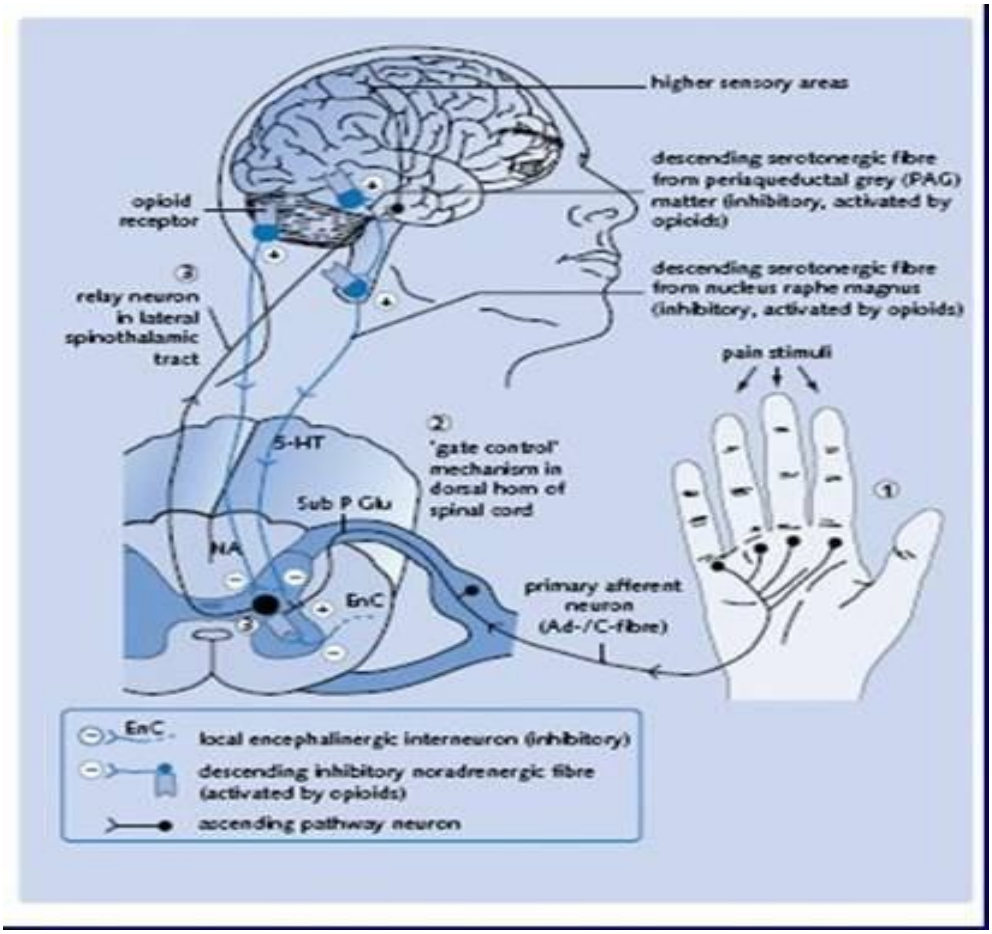
ENDOGENOUS INHIBITORY MECHANISMS:

Spinal mechanisms:

Melzack and Wall proposed the "Gate control theory". It postulates that there is selective activation of non-nociceptive afferent Aβ fibres which will recruit inhibitory interneurons in the substansia gelatinosa producing a localized analgesia and decreased pain perception.

Diffuse noxious inhibitory controls:

Periaqueductal grey matter and NRM are important seratonergic and noradrenergic inhibitory pathways. There will recruit encephalinergic interneurons in spinal cord to produce analgesia.



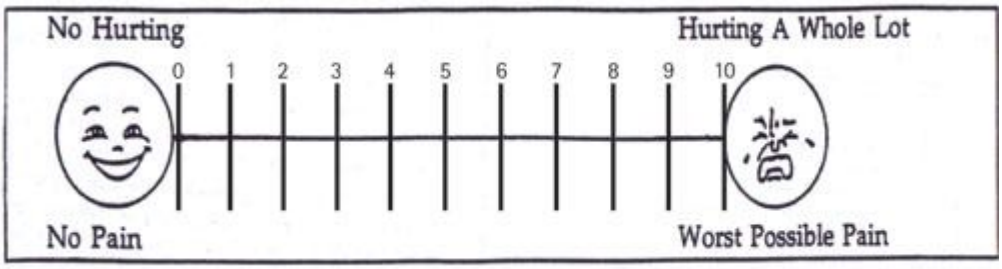
Superior control centers:

Cognitive manipulations like distractions and hypnosis can influence pain perception. They will alter both the affective and sensory components of pain perception.

PAIN RATING SCALE

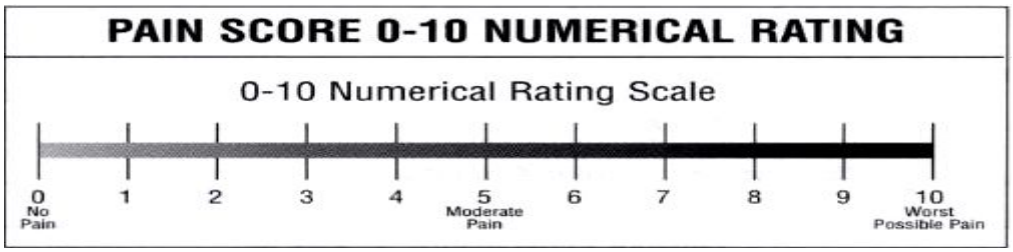
Visual analogue scale:

Instructions: Please mark on the line how you felt about this injection.



The patient is instructed to point a position on the line between the faces to indicate how much pain they are experiencing. The left end denotes 'No pain' and the right end denotes 'Worst pain ever'.

Numerical rating scale (NRS)



The patient is instructed to choose a number from 0 to 10 that best describes their current pain. 0 denotes 'No pain' and 10 denotes 'Worst possible pain'.

Faces Rating Scale (FRS)

Adults having difficulty in using the numbers on the visual/numerical rating scales can be assisted with the use of this six facial expressions suggesting a range of pain intensities. Ask the patient to choose the face that describes their pain. The extreme left face denotes 'No hurt' and the extreme right face denotes 'Hurts worst'.

Behavioural rating scale:

Patients who are unable to provide a self-report of pain this scale can be used: scored 0–10 clinical observation

Face	**0** Facial muscles relaxed	**1** Facial muscle tension, grimace	**2** constant grimace, clenched jaw	**Face score:**
Restlessness	**0** Quiet, relaxed appearance, normal movement	**1** Occasional agitated movement	**2** Frequent agitated movement including extremities or head	**Restlessness score:**
Muscle tone*	**0** Normal muscle tone	**1** Increased tone, flexion of fingers and toes	**2** Rigid	**Muscle tone score:**
Vocalisation**	**0** No abnormal sounds	**1** Occasional moans, cries and grunts	**2** Frequent or continuous moans, cries or grunts	**Vocalisation score:**
Consolability	**0** Content, relaxed	**1** Can be reassured	**2** Difficult to reassure	**Consolability score:**
Behavioural pain assessment scale total (0–10)				**/10**

The behavioural pain assessment scale is intended for use with non-verbal patients unable to expresss self-reports of pain.

Functional activity score:

This is an activity-related score. Ask the patient to do an activity associated to their painful area like cough or movement.

A – No limitation

B – Mild limitation

C - Severe limitation

OPIOID RECEPTORS[8],[9],[13],[23],[25]

Opioid receptors are expressed widely both in the central as well as peripheral nervous system. There are mainly three opioid receptors namely mu, kappa and delta opioid receptor.

STRUCTURE AND FUNCTION:

Opioid receptors are G-protein coupled receptors and they possess 7 alpha helical transmembrane domains with intracellular loops and extracellular N terminus. The difference between the receptors lie in the N and C terminus as well as the extracellular loops.

When the receptor gets activated, G-protein coupling occurs, causing activation of the inward rectifying potassium channels, inhibition of adenylyl cyclase and voltage dependant calcium channels. Alteration in potassium and calcium conductance leads to decrease in neuronal excitability and inhibition of neurotransmitter release.

Opioid receptors mediate antinociception by variety of mechanisms. These include,

1) Decreasing the Ca^{2+} current by inhibiting high voltage activated Ca^{2+} channels seems to be the primary mechanism for the inhibiting effects of opioids on primary afferent neurons.

2) Inhibit Ca^{2+} dependent substance P release from peripheral sensory nerve endings.

3) Activation of inward rectifying K+ channels causes decrease in neuronal excitability.

Peripheral opioid receptor expression:

In addition to spinal and centrally placed opioid receptors, they are also expressed in peripheral neurons which contributes to antinociception.

In the peripheral neurons, opioids produce their effect by local activation of adenosine tri phosphate(ATP) sensitive potassium channels and inhibition of L-type calcium channels.

Opioid receptors are produced in dorsal root ganglia (DRG) and transported by axonal transport to both central and peripheral tissues. It is postulated that, during inflammation there is significant increase in opioid receptors both in dorsal root ganglion and peripheral nerves. The mechanism behind these increase in receptors is increase in IL-6 in inflamed tissues which induces opioid receptor transcription and translation. Also the proinflammatory cytokine Tumour necrosis factor induces opioid receptor gene transcription.

There is increased opioid receptor expression due to alteration in subcellular distribution of receptor and disruption of perineural barrier thereby providing opioids, a better access to the receptors. Therefore, administration of peripheral opioid peptides provide antinociception and limits the systemic side effects.

APPLIED ANATOMY[12]

BRACHIAL PLEXUS:

The effective use of brachial plexus blockade for upper limb surgeries needs thorough knowledge about the anatomy of the brachial plexus. It is essential to know about the formation, distribution, its vascular, muscular and fascial relationships to master this technique. The fibres that constitute the plexus are composed of roots, trunks, cords, divisions and terminal nerves.

FORMATION OF THE PLEXUS

ROOTS

The plexus is formed by the anterior primary rami of 5^{th} to 8^{th} cervical nerves and 1^{st} thoracic nerve. Occasionally 4^{th} cervical nerve is combined with the plexus and called as prefixed plexus (C4-C8) or second thoracic nerve is combined with plexus and called as postfixed plexus (C6-T2).

TRUNKS

The roots emerge from the intervertebral foramina and lie between the anterior and posterior tubercles of the respective transverse process. As the roots descend between the scalenus anterior and medius, C5 and C6 roots unite to form the upper trunk. C7 root continues as the middle trunk and C8 and T1 unit to form the lower trunk. Each trunk divides into anterior and posterior divisions behind the clavicle and form cords in the axilla.

CORDS

The stream of six divisions join up into three cords; lateral, medial and posterior and are composed of as follows. Anterior divisions of upper and middle trunks unite to form the lateral cords. Anterior divisions of the lower trunk continues as the medial cord posterior divisions of upper, middle and lower trunks unite to form the posterior cord.

To summarise the formation of brachial plexus,

1) Five roots- Ventral primary rami of C5-C8 and T1.

2) Three trunks

   a. Upper trunk C5-C6

   b. Middle trunk C7

   c. Lower trunk C8-T1

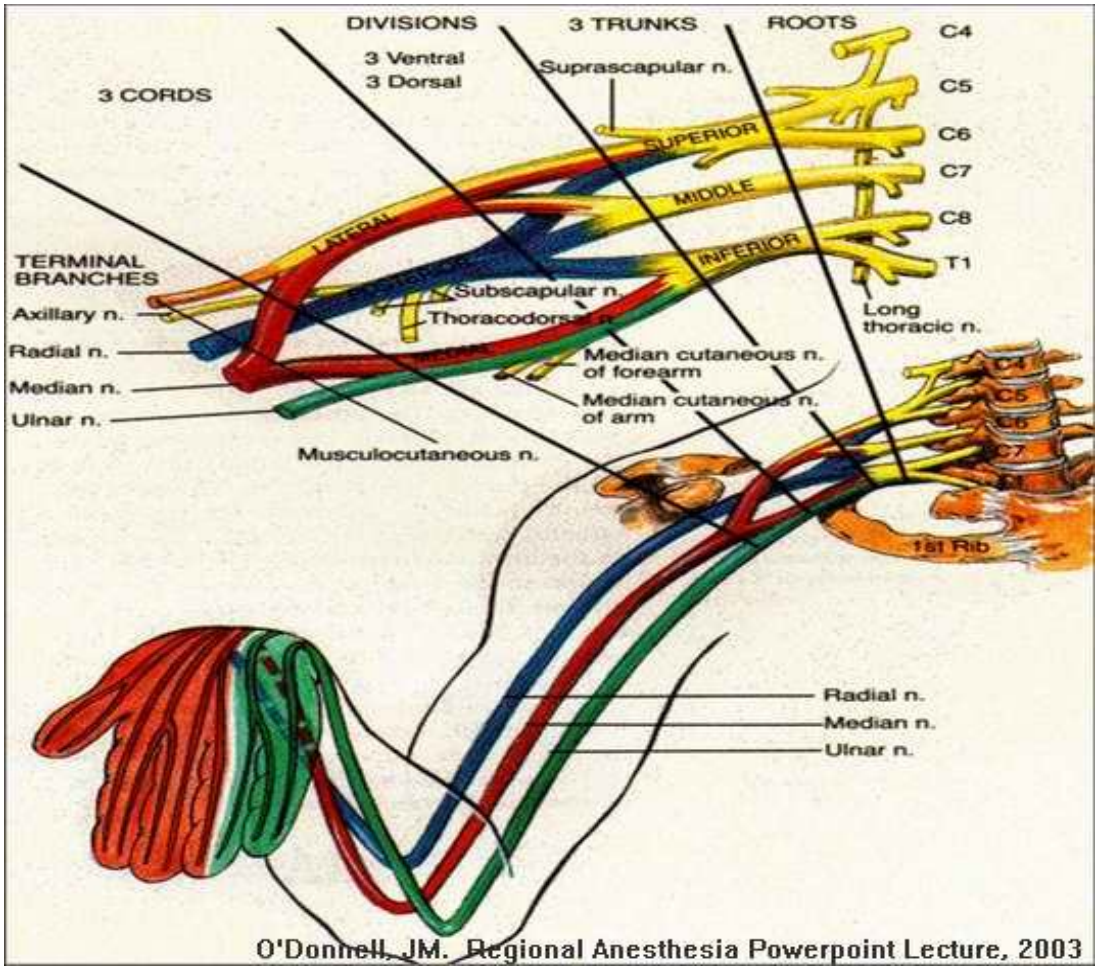
3) Six divisions – each trunk divides into anterior and posterior divisions.

4) Three cords

   a. Lateral cord- the fused anterior divisions of upper and middle trunk C5-C7.

   b. Medial cord- anterior division of the lower trunk C8-T1.

c. Posterior cord- formed by union of posterior divisions of all three trunks (C5-T1).



RELATIONSHIP OF THE BRACHIAL PLEXUS :

ROOTS

Lie between the scalenus anterior and medius muscle and above the 2nd part of subclavian artery.

TRUNKS

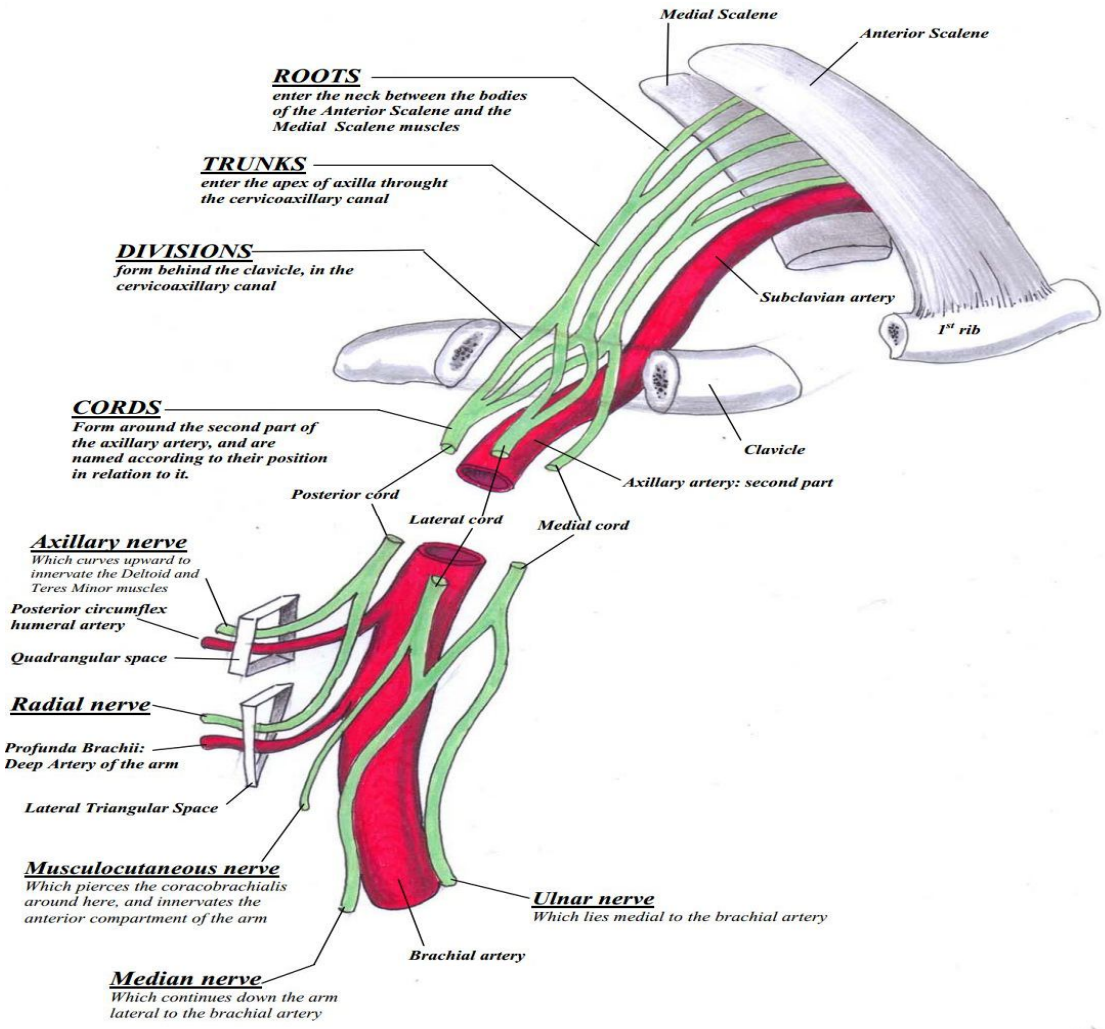
Upper and middle trunks lie above the subclavian artery but lower trunk behind it as they cross the first rib.

DIVISIONS

At the lateral border of the first rib the trunks give rise to divisions behind the clavicle.

CORDS

Formed at the apex of the axilla and grouped around the axillary artery.



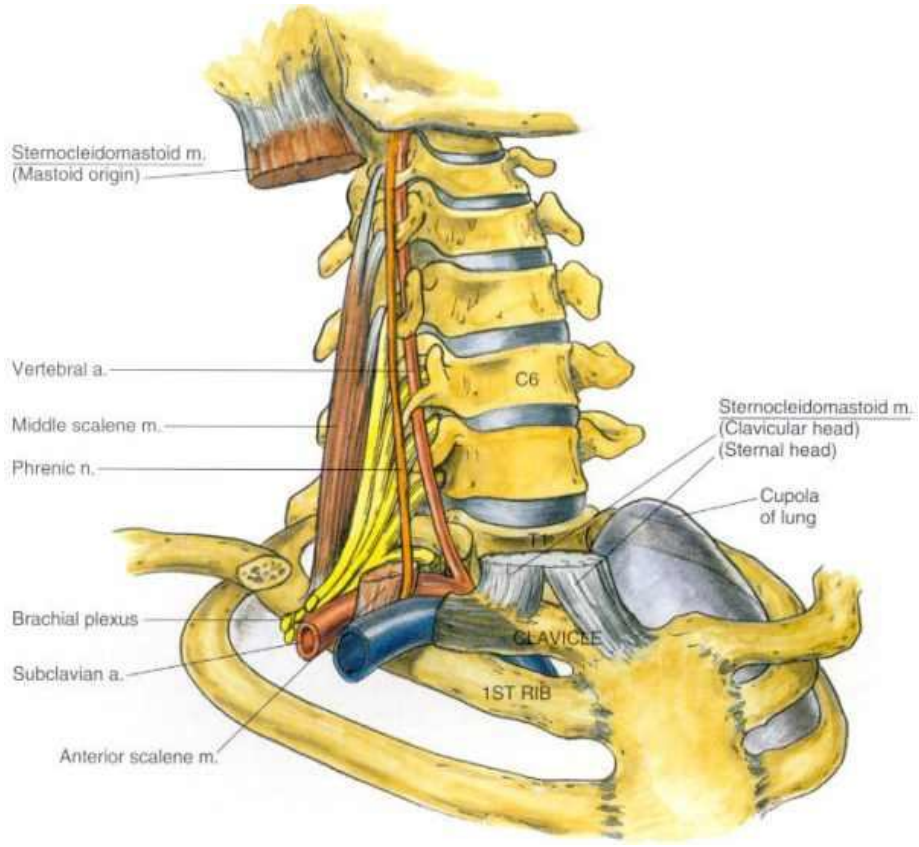
SYMPATHETIC SUPPLY

5^{th} and 6^{th} cervical nerves each receive a grey ramus from the middle cervical sympathetic ganglias. 7^{th} and 8^{th} cervical nerves receive from the inferior cervical ganglion.

BLOOD SUPPLY

The cord and rootlets are supplied by the anterior and posterior spinal branches of the vertebral artery. Trunks are supplied directly and indirectly by muscular branches of the ascending deep cervical and superior intercostal arteries.

Cords also receive small branches from subclavian, axillary and subscapular vessels.



BRANCHES

Branches are given off from roots, trunks and cords.

BRANCHES FROM ROOTS

1) Nerve to the serratus anterior C5, C6 and C7.

2) Muscular branches to

   a. Longus cervices C5-C6

   b. Three scalene muscles C5-C8

   c. Rhomboids C5

3. Twig to the phrenic nerve C5

BRANCHES FROM THE TRUNKS

1) Suprascapular nerve C5-C6 (Upper trunk)

2) Nerve to subclavius C5-C6

BRANCHES FROM THE CORDS

Lateral Cord

- Lateral pectoral nerve C5-C7
- Lateral branch of median nerve C5-C7
- Musculocutaneous nerve C5-C7

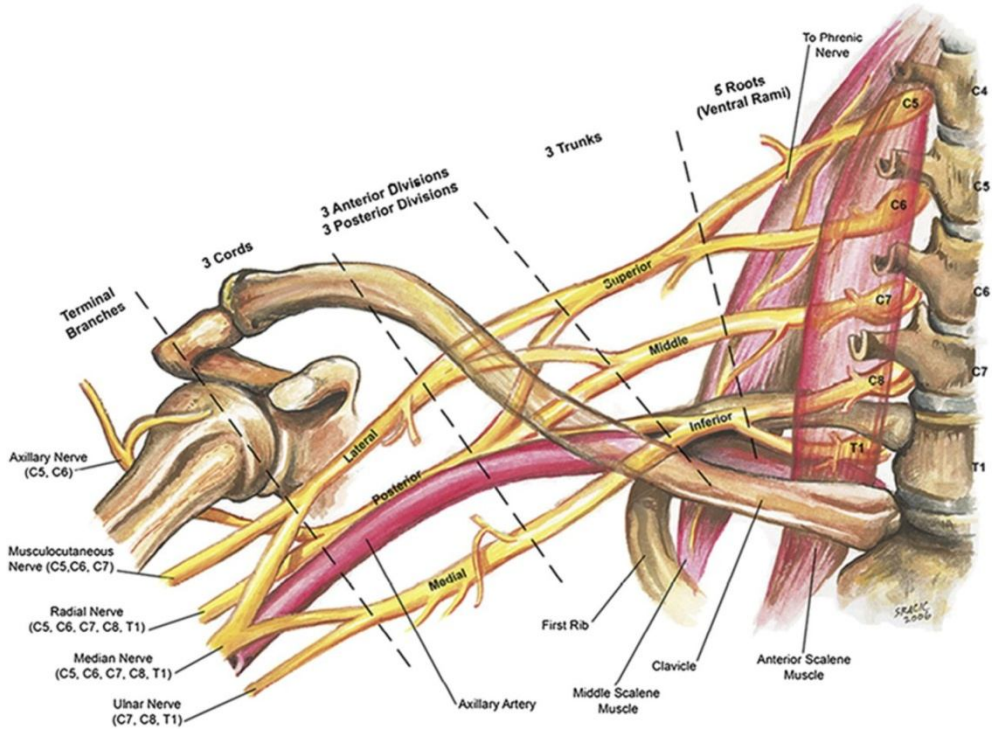
Medial Cord

- Medial pectoral nerve C8-T1
- Medial branch of median nerve C8-T1
- Medial cutaneous nerve of arm C8-T1
- Medical cutaneous nerve of forearm C8-T1
- Ulnar nerve (C7, C8, T1)

Posterior Cord

- Upper subscapular nerve (C5-C6)
- Lower subscapular nerve (C5-C6)

- Nerve to lattismus dorsi C6, C7, C8
- Axillary nerve C5-C6
- Radial nerve C5-T1



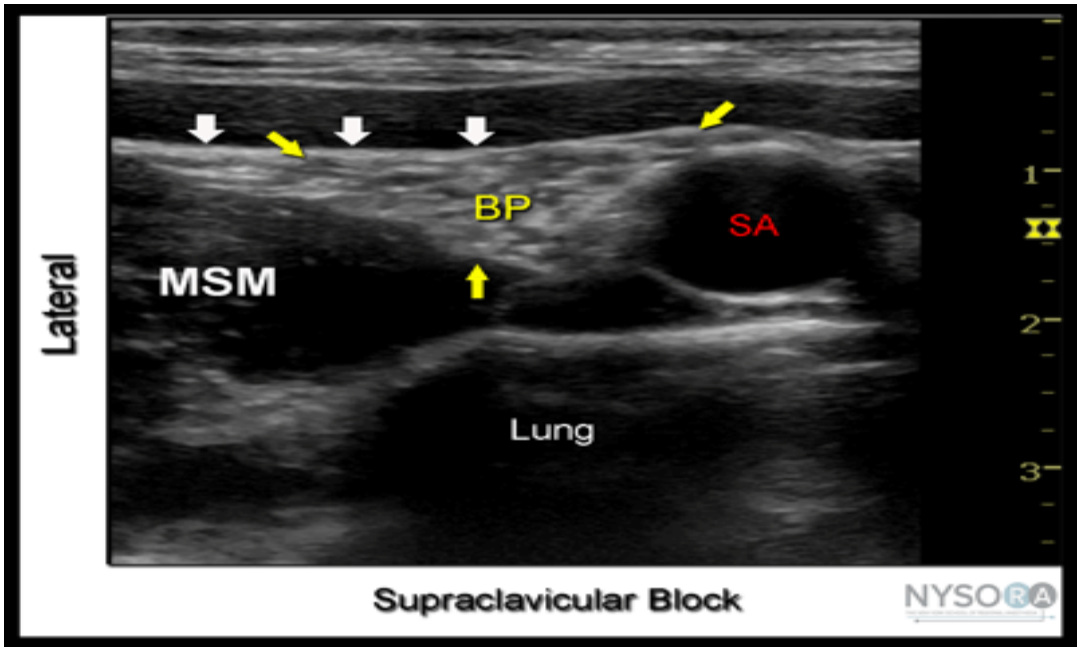
SONOANATOMY OF BRACHIAL PLEXUS[(10)]

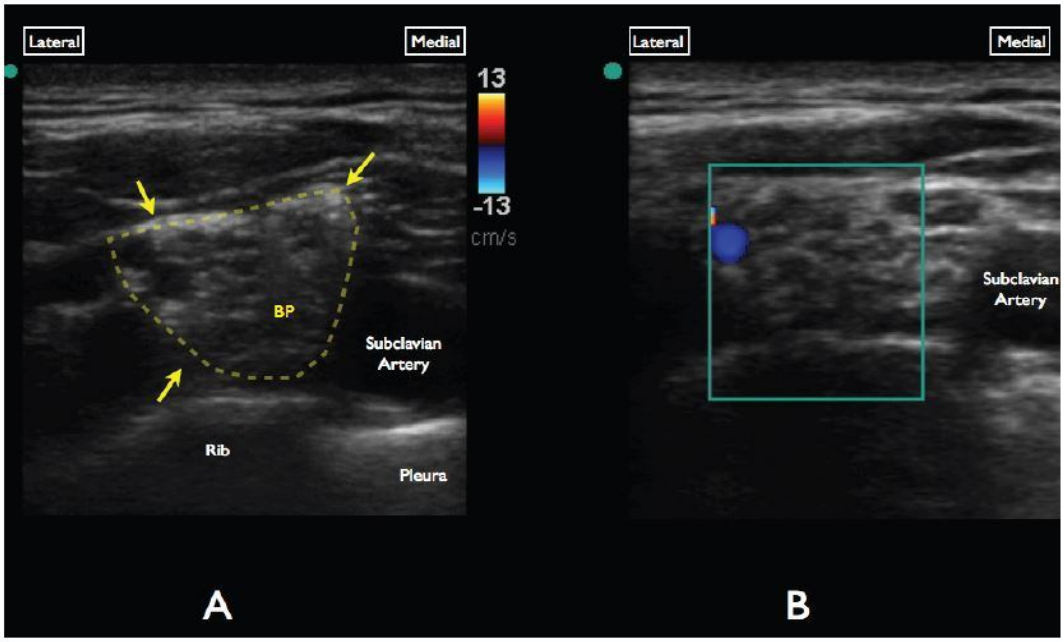
The landmark to be identified above the first rib is subclavian artery which is seen as a prominent pulsatile hypoechoic structure which resembles like tennis ball.

First rib appear as a bright hyperechoic structure with a bony acoustic shadow.Brachial plexus appears as multiple hyperechoic ovals or circles superior or superolateral to the subclavian artery. The appearance is often described as “honey comb pattern” or “bunch of grapes”.

The pleura is seen at the same level as rib as hyperechoic line and differentiated by the absence of drop out acoustic shadow.

The smaller artery pulsations are masked by the stronger subclavian artery pulsation. This poses threat of injury to these vascular structures, hematoma formation and intravascular injection. This can be prevented by using colour flow Doppler during the block.





TECHNIQUES OF BRACHIAL PLEXUS BLOCK[5],[14],[20]

Brachial plexus can be blocked at various sites by various approaches as follow

1) Interscalene approach

2) Supraclavicular approach

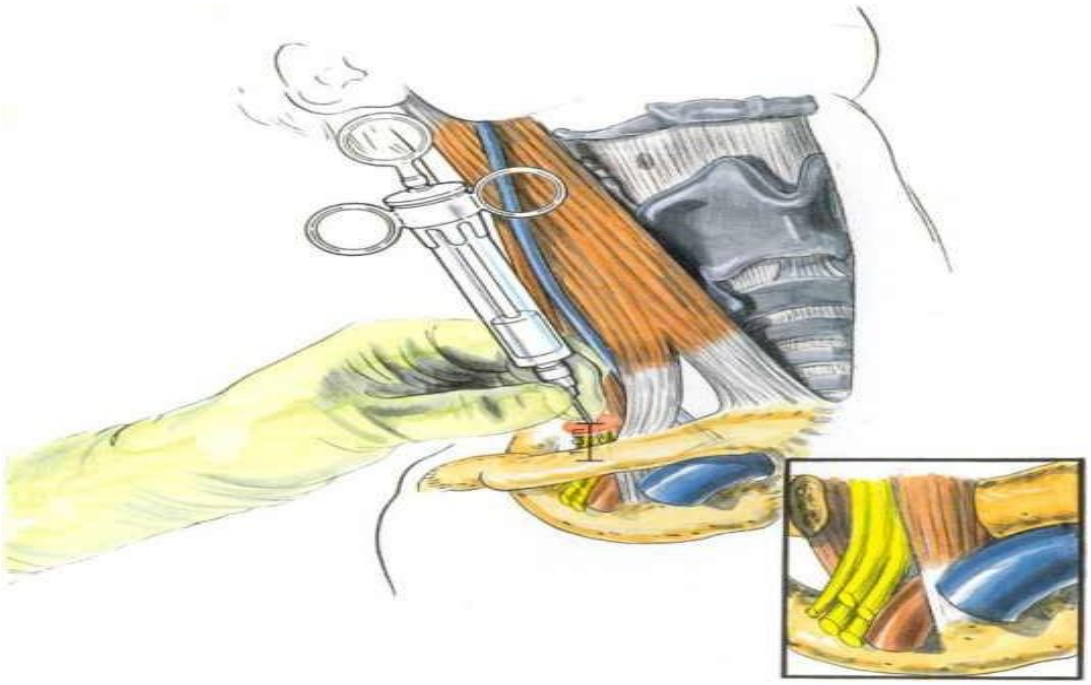
3) Axillary approach

4) Infraclavicular approach

SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK

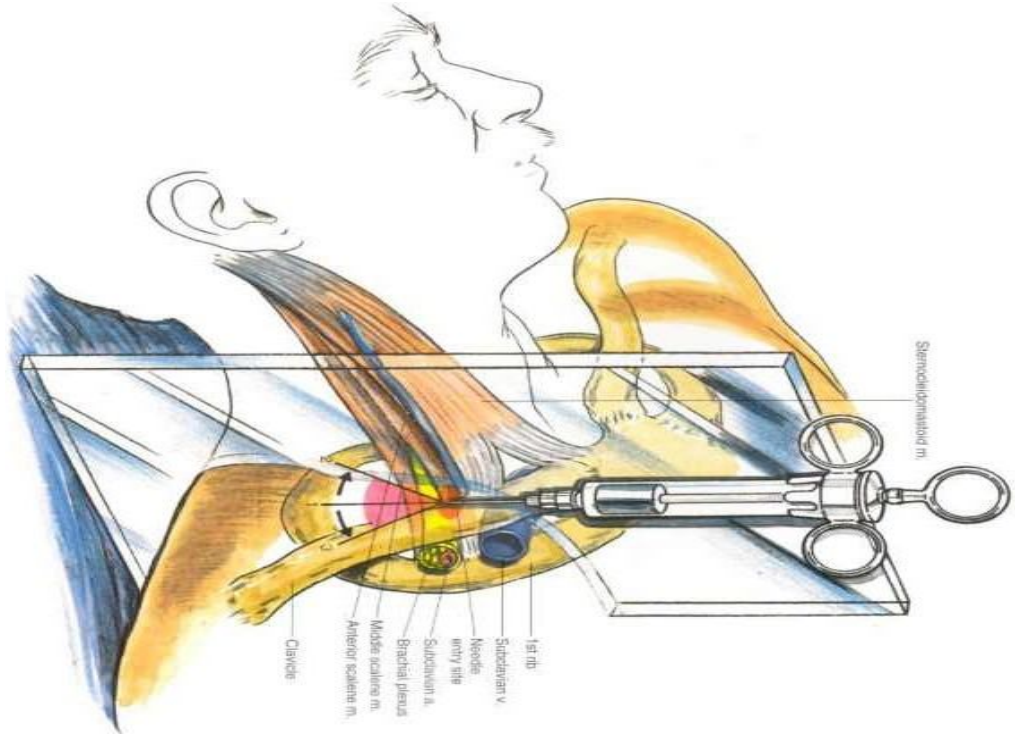
CLASSIC KULENKAMPFF APPROACH

In this, the needle insertion point is marked at 1cm superior to the clavicular mid point. The needle is inserted parallel to the patient's head and neck. Once the needle contacts the rib at a depth of 3 to 4cm, the needle is walked over the rib for paraesthesia. Local anaesthetic solution is given after eliciting parasthesia after careful negative aspiration.



PLUMB BOB TECHNIQUE

The needle insertion point is marked superior to the clavicle where the sternocleidomastoid muscle gets inserted into the clavicle. The needle is inserted at 90^0 angle to the table. The local anaesthetic solution is given after eliciting paraesthesia.



SUBCLAVIAN PERIVASCULAR TECHNIQUE

The intercalene groove is palpated and trace it below to the most inferior part which is just posterior to the subclavial artery pulsation. The needle is entered just above and posterior to the arterial pulse and directed caudally at a flat angle with the skin. Once the paraesthesia is elicited, local anaesthetic is given.

COMPLICATIONS

1) Pneumothorax

2) Horner's Syndrome

3) Phrenic nerve blockade

4) Hemothorax

5) Hematoma Formation

ULTRASOUND GUIDED TECHNIQUE[10],[17],[19]

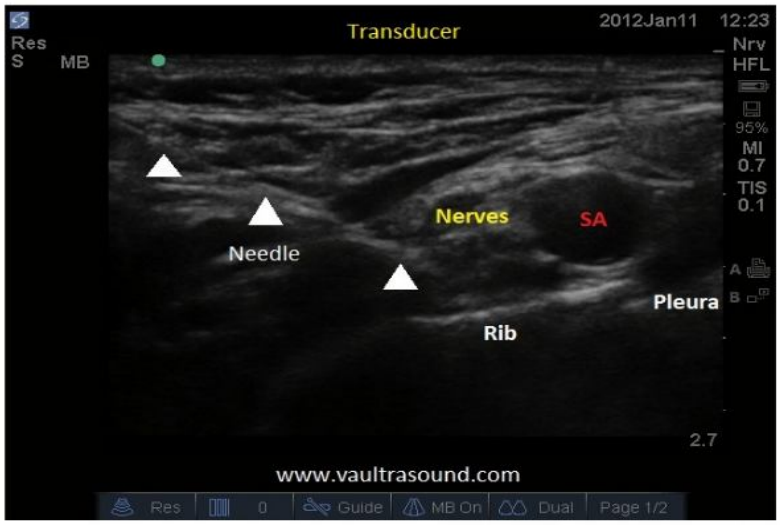
Here, high frequency ultrasound transducer is used to visualise the superficial structures.

Ultrasound probe is placed in the supraclavicular fossa in a coronal oblique plane. The supraclavicular artery pulsation is noted above the hyperechoic first rib. The probe is then angled slightly until the 1st rib and pleura are simultaneously seen. The hypoecheic nerve structures are visualized superolateral to the artery. Then using "Inplane" technique, the

needle is inserted in a lateral to medial direction and guide the needle towards the “corner pocket” formed by 1st rib inferiorly, subclavian artery medially and the brachial plexus superiorly. Here, lies the lower trunk of plexus and failure to block this area results in inadequate ulnar nerve anaesthesia.

Local anaesthetic spread during injection is observed and the needle can be repositioned to ensure drug distribution all around the nerve trunks within the plexus.

Throughout the procedure, the needle tip should be visualized. If the tip is not visible, hydrodissection technique can be used to locate the tip of the needle.



Advantages of Ultrasound Guidance [29]

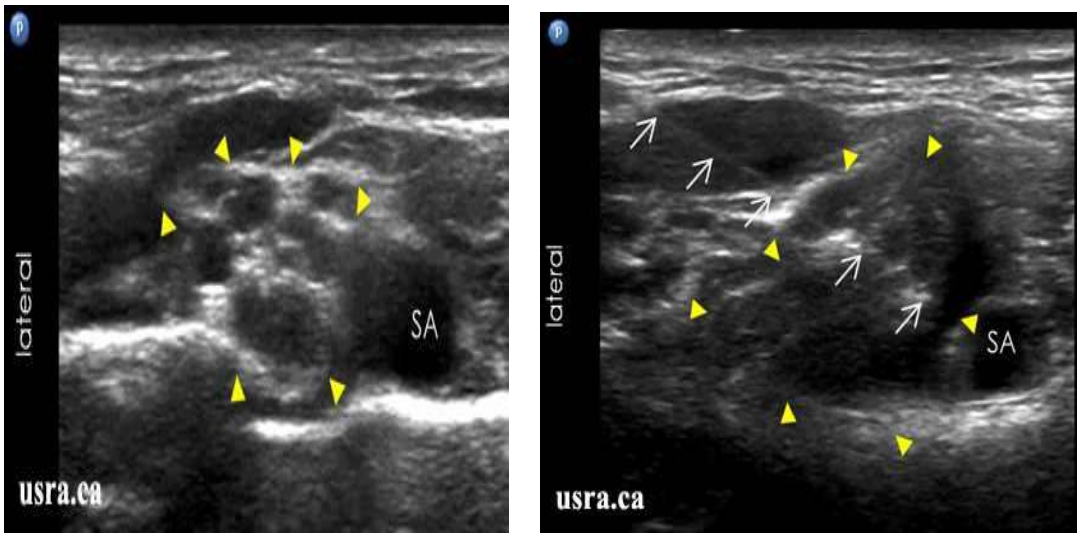
Ultrasound guidance with real-time needle visualization in relation to anatomic structures and target nerves makes regional anesthesia safer and more successful.

With ultrasound guidance, brachial plexus blockade can lead to

- Decreased block performance and onset time,
- Increased success rate and
- Decreased rate of complications.

These advantages result in increased operating room efficiency, as well as increased patient satisfaction.

Preinjection: Postinjection



PHARMACOLOGY OF BUPIVACAINE[1],[22],[31]

Bupivacaine hydrochloride is 1-butyl 2^1, 6^1 Pipecoloxylilide hydrochloride. It is a synthetic long acting amide local anaesthetic. EKENSTAM first synthesized bupivacaine in 1957 at A.B. Bafors Laboratories in Mo Indel, Sweden.

PHYSIOCHEMICAL PROPERTIES

Bupivacaine has a butyl group on the piperidine nitrogen atom and is a long acting drug with high anaesthetic potency. It is highly protein bound and has high lipid solubility. It is 3 to 4 times potent as lignocaine. It crosses both placenta and blood brain barrier.

1) Molecular weight – 288.42 g/mol

2) pKa – 8.1

3) Partition Coefficient- 346

4) Mean uptake ratio - 3.3

5) Protein binding- 95 %

PHARMACOLOGICAL PROPERTIES

1) Onset- slow

2) Duration- Long acting (4- 8 hours)

MECHANISM OF ACTION

Bupivacaine produces electrical stabilization of the membrane by action on sodium conductance.

Inhibiting the conformational change in the membrane by binding with inactivated state of Na+ Channel, thereby preventing the depolarization.

PHARMACOLOGICAL EFFECTS

Local	:	Nerve blockade
Regional	:	Pain, temperature, touch, motor , power and vasomotor tone in the region supplied by nerves that are blocked.
Systemic	:	Effects occurring as a result of systemic absorption or IV administration

On the cardiovascular system, the effect of bupivacaine is dose related. At higher concentration, it blocks sufficient cardiac Na+ channels causing conduction blockade, decreased myocardial contractility and depressed Vmax (maximum depolarization rate of action potential) leading to slower conductance. This is manifested as prolonged PR interval and QRS interval on ECG. This results in reentrant phenomenon and ventricular arrhythmias.

Bupivacaine block cardiac Na+ channels during systole. Whereas during diastole it dissociates off these channels at much slower rate than lignocaine because of high lipid solubility causing persistent Vmax

depression and subsequent cardiac toxicity. This causes difficulty in resuscitation when cardiotoxicity occurs.

PHARMACOKINETICS

Volume of distribution	:	73 Litres
Terminal Elimination half life	:	210 Minutes
Clearance	:	0.58L/ Min
Plasma Protein binding	:	98%
Metabolism	:	Liver by Dealkylation to pipecolyloxilidine.
Excretion	:	5% by the kidney as unchanged drug and the rest as metabolites

PREPARATION AVAILABLE:

0.125%

0.25%

0.5%

PHARMACOLOGY OF BUPRENORPHINE[2],[23],[24]

Buprenorphine is a semi synthetic highly lipophilic agonist antagonist opioid derived from the opium alkaloid thebaine. It is 33 times more potent than morphine.

MECHANISM OF ACTION

It is a partial agonist at μ receptor. Its affinity to μ receptor is 55 times greater than that of morphine. It also binds to delta and kappa receptors and acts as antagonist. Buprenorphine binds with high affinity and also blocks voltage gated Na channels and this leads to local anaesthetic property of buprenorphine.

PHARMACOKINETICS

Buprenorphine undergoes extensive first pass metabolism and has low oral bioavailability. But its bioavailability is extensive in sublingual route. Administered sublingually drug produces satisfactory analgesia. The time to achieve maximum plasma concentration is 40 minutes to 3.5 hours when given sublingually or orally whereas 5 minutes after IM injection.

Its peak effect may take upto 3 hours and duration upto 10 hours. The drug remains in the tissues for several days. Elimination half life is 24 - 60 hours.

Since it is highly lipophilic, its association and dissociation from the receptor is very slow. Half life for dissociation is 166 minutes. Compared to 7 minutes for fentanyl. So plasma levels may not parallel clinical effects.

Protein binding- 96%

Volume of distribution -2.8L

Plasma clearance - 20ml / Kg

Metabolism: Liver by dealkylation and conjugation to norbuprenorphine and buprenorphine- 3 glucuronide through CYP3A4. One of the major active metabolites is norbuprenorphine which is a full agonist at mu receptor and a partial agonist at kappa receptor. But it has $1/50^{th}$ of antinociceptive potency and 10 times that of respiratory depressive potency when compared to buprenorphine. Buprenorphine 3 glucuronide and norbuprenorphine 3 glucuronide are also biologically active. Buprenorphine 3 glucuronide has affinity for the mu recceptor and delta receptor but no affinity for kappa receptor. Norbuprenorphine 3 glucuronide has no affinity for mu and delta receptor but bind to kappa opioid receptor and produces sedative effect but do not depress the respiration.

Excretion- Most are excreted in bile through feces and 10-35% in urine.

Therefore, Pharmacokinetics are not much altered in patients with renal impairement.

INTERACTIONS:

1) CYP3A4 inhibitors – buprenorphine actions will get potentiated when used along with the drugs like azole antifungal agents, macrolides, ART drugs.

2) CYP3A4 inducers – buprenorphine actions will be decreased by the drugs like phenobarbitonr, phenytoin, benzodiazepines, carbamazepine, opioid analgesics, general anaethetic drugs, phenothiazones, sedative hypnotics, alcohol and other CNS depressant drugs.

CONTRAINDICATIONS:

1. Allergic to the drug
2. Severe respiratory insufficiency
3. Severe hepatic impairment
4. Acute alcoholism or delirium tremens

PHARMACOLOGICAL ACTIONS:

CARDIOVASCULAR SYSTEM:

Buprenorphine produces vasodilatation and a decrease in heart rate and blood pressure. Postural hypotension is prominent. Pulmonary edema has been reported.

CENTRAL NERVOUS SYSTEM:

It produces significant respiratory depression with a ceiling effect after doses of 0.15 to 1.2mg. Increased doses do not produce further depression and may actually result in increased ventilation due to antagonistic action. Because of the high affinity and slow dissociation from the receptor its reversal is limited. High doses of naloxone are required for reversal of respiratory depression. In the epidural space, the high lipid solubility limits the cephaled spread of the drug and likelihood of delayed respiratory depression than morphine.

Sedation, drowsiness, miosis, nausea and vomiting are similar to morphine. Dysphoria is unlikely. constipation is less prominent than morphine.

ADVERSE EFFECTS:

CNS:

- Headache
- Migraine
- Drowsiness
- Somnolence
- Miosis

RESPIRATORY SYSTEM:

- Respiratory depression
- Cough

GASTROINTESTNAL SYSTEM:

- Constipation
- Nausea
- Vomiting
- Abdominal pain
- Loose stools
- Dyspepsia

SKIN:

- Pruritis
- Rash

MUSCULOSKELETAL SYSTEM:

- Arthralgia
- Myalgia
- Muscle spasm

PSYCHIATRY:

- Anxiety
- Depression
- Insomnia nervousness

TOLERANCE AND DEPENDANCE

There is lower degree of tolerance to buprenorphine. Physical and psychological dependence occur with this drug compared to morphine on chronic use.

USE IN OBSTETRICS

Not recommended during labour because of respiratory depression occurs in the neonate and cannot be reversed with naloxone.

USE IN PEDIATRICS:

Safety not evaluated

DOSAGE IN ADULTS:

Sublingual – 0.4-0.8mg

IV/IM: 0.3mg

Intrathecal, epidural and Peripheral nerve blockade- 2 to 4 μg/Kg

USES:

1) As an analgesic for long lasting painful conditions like cancer pain.

2) For control of postoperative pain.

3) As an analgesic component of balanced anaesthesia (4.5-12μg/Kg)

4) For intraoperative and postoperative analgesia by intrathecal, epidural and also through peripheral nerve blocks.

5) As a maintenance drug for opioid depression patients as an alternative to methadone.

REVIEW OF LITERATURE

Regional anaesthetic technique are widely used for orthopaedic procedures because of excellent intraoperative and post operative analgesia. But, local anaesthetics above cannot provide post operative analgesia for a longer duration.

To overcome this, several adjuvants have been studied along with local anaesthetics to prolong the duration of post operative analgesia without much side effects.

Opioids as adjuvants have been studied for prolongation of postoperative analgesia. The mechanism of action postulated was

1) There would be movement of opioids to their receptors through an axoplasmic flow.

2) Other would be that of a diffusion and subsequent binding to the receptors of lamina V in the spinal cord.

But, Now it has been demonstrated that, peripheral afferent sensory fibres contain opioids receptors, in addition to that endogenous opioids also been demonstrated in the peripheral sensory fibres.

SUPRACLAVICULAR APPROACH FOR BRACHIAL PLEXUS BLOCK

1. Winnie and Ramamoorthy (1977) postulated that the trunks of brachial plexus are arranged into 2 groups, the peripheral mantle and central

core fibres. The mantle bundle contains the outer motor and inner sensory fibres. The core bundle contains outer motor supplying the muscles of forearm and inner sensory fibres carrying sensations from the hand.

Thus, the order of blockade is as follows loss of motor power of shoulder and upper arm, loss of sensation in upper arm, loss of motor power of the forearm and loss of sensation of the hand.[30]

2. Brown DL et al (1993) did a study on various sites of brachial plexus block. They studied the supraclavicular, interscalene, infraclavicular and axillary approaches. They conducted that the supraclavicular block produces anaesthesia of the entire upper extremity in an efficient manner than any other technique. This is due to arrangement of plexus in a very compact manner in this site.[5]

ULTRASOUND GUIDED TECHNIQUE FOR BRACHIAL PLEXUS BLOCK

1. Vienna et al (1994) developed the ultrasound guided technique for the supraclavicular approach and demonstrated the high success rate with this technique

2. William et al (2003) compared the ultrasound guided supraclavicular brachial plexus block with nerve stimulation technique and found that ultrasound guided was superior than nerve stimulator technique.[29]

3. Yuan Jia Min et al (2012) studied the complications of ultrasound guided and peripheral nerve stimulator guidance brachial plexus block and he

found out that ultrasound guided technique decreases the risks of vascular puncture and improves the success rate of brachial plexus compared with peripheral nerve stimulator technique

4. Krutika et al (2013) studied the usefulness of ultrasound guided supraclavicular brachial plexus block and compared it with nerve stimulator guided technique. Group I – USG guided technique were used and Group –II nerve stimulator technique were used. They compared block execution time, time of onset of sensory and motor blockade, quality of blockade and success rates. From this study they concluded that USG guided technique is quick to perform with improved safety in positioning the needle and accuracy in visualizing the anatomical structures.[16]

PERIPHERAL ANTINOCICEPTIVE ACTIONS OF OPIOIDS

1. Hassan et al (1993) have showed that the axonal transport of opioids receptors in sciatic nerves is increased in peripheral inflammation with increase in number of opioids receptors on peripheral nerve fibres.[13]

2. Stein et al (1997) demonstrated that peripheral opioid receptors are present on peripheral sensory nerve terminals and characteristics are similar to those in the brain. Antinociceptive effects of opioids is due to the inhibition of calcium dependent release of substance P from spinal cord.[25]

3. Karakaya et al (2001) did a study comparing the analgesic efficacy of fentanyl when added with bupivacaine for axillary brachial plexus block. They concluded that addition of fentanyl almost doubles the duration of analgesia when compared with bupivacaine alone.[15]

4. Jena et al (2013) did a study to evaluate the effect of butorphanol versus placebo as adjuvant to bupivacaine for supraclavicular brachial plexus blockade on the onset of blockade and duration of analgesia. They calculated that butorphanol prolongs the duration of blockade and increase the postoperative analgesia without much adverse effects.

BUPRENORPHINE WITH LOCAL ANAESTHETICS IN BRACHIAL PLEXUS BLOCK

1. Viel EJ et al (1997), studied the efficacy of buprenorphine and morphine in supraclavicular brachial plexus block. They noted that there is significant difference in duration of analgesia with buprenorphine group had two fold greater analgesia than the morphine group.[27]

2. Kenneth D. Candido et al (2002), conducted a study to evaluate the efficacy of buprenorphine in prolonging the analgesia when added to local anaesthetic in subclavian perivascular technique. They have concluded that there is three fold increased duration of analgesia in buprenorphine group patients when compared to patients given local anaesthetic alone.(6)

3. Winnie et al (2002), conducted a study comparing buprenorphine added to local anaesthetic for axillary brachial plexus block versus local anaesthetic alone with regards to postoperative analgesia.

   Group I received local anaesthetic with buprenorphine in axillary block and intramuscular saline, Group II received local anaesthetic alone in axillary block and intramuscular buprenorphine and Group III received local anaesthetic alone in axillary block and intramuscular saline.

   Duration of analgesia was compared and concluded that postoperative analgesia in Group I was 3 times prolonged than Group III and 2 times prolonged than Group II and supported the peripheral action of opioids.[30]

4. Dhrubajyothi Sarkar et al (2010), evaluated the effects of adding fentanyl and buprenorphine to local anaesthetics in brachial plexus block by comparing 3 groups using subclavian perivascular technique. Group-I received the block with local anaesthetic alone. Group-II received the block with local anaesthetic and 0.3mg buprenorphine. Group-III received the block with local anaesthetic and 50μg fentanyl. They conducted that the addition of buprenorphine to local anaesthetics provides a significant prolongation of analgesia than local anaesthetics alone and local anaesthetics with fentanyl with no significant adverse effects.

5. Singam et al (2012), did a study in which Group-C received local anaesthetic alone and Group-B received local anaesthetic and

buprenorphine 0.3mg. They concluded that buprenorphine, as adjuvant to bupivacaine in brachial plexus block prolonged the duration of analgesia without increasing the risk of adverse effects and is excellent choice for providing postoperative analgesia.[2]

6. Bharat et al (2013), conducted a prospective randomized double blinded study and compared the effects of buprenorphine and clonidine as adjuvants to local anaesthetic 0.25% bupivacaine for supraclavicular brachial plexus block.

   Group B received 0.5% bupivacaine 20 ml + 20 ml NS
   Group BB received 0.5% bupivacaine 20 ml +0.3mg buprenorphine +19 mlNS
   Group BC received 0.5% bupivacaine 20 ml + 150µg clonidine + 19 mlNS

   They conclude that Vas score were lower in group with buprenorphine than without it at all times except at 24 hour at which the three groups showed no difference between them. Also the time for first rescue analgesic was significantly prolonged in buprenorphine (18±6.391)and clonidine group (15.3±4.78) than plain bupivacaine group (9±2.791) .[4]

7. Ritesh et al (2013), conducted a study on effect of buprenorphine in supraclavicular brachial plexus block on postoperative analgesia. Group I received 0.25% bupivacaine 40 ml and Group II received 0.25% bupivacaine and 3 microgram/kg buprenorphine. Onset of action,

duration of analgesia, time of first rescue analgesia and number of analgesic drug required in the first 24 hours were studied.

They concluded that use of buprenorphine in brachial plexus block prolongs postoperative analgesia by more than 2 fold.[21]

8. Deepali et al (2015), conducted a prospective randomized double blinded placebo controlled study and compare the effect of buprenorphine with local anaesthetic in axillary brachial plexus block versus intramuscular buprenorphine.

   Group RB received buprenorphine 2 microgram/kg with local anaesthetic in axillary block and 1ml NS intramuscularly. Group SB received 1ml NS with local anaesthetic in axillary block and buprenorphine 2 microgram/kg intramuscularly.

   They concluded that patients who have received buprenorphine in axillary block had significantly prolonged analgesia when compared to the patients who have received intramuscular buprenorphine.

MATERIALS AND METHODS

This study was carried out in the orthopaedic theatre, Government General Hospital, Chennai after obtaining Institutional approval between Jan 2015 to June 2015. The aim of the study was to evaluate the efficacy of Buprenorphine as an adjuvant to bupivacaine for ultrasound guided supraclavicular brachial plexus block in patients undergoing elective upper limb surgeries.

STUDY DESIGN

The study was a prospective randomized control study.

SELECTION OF CASES

60 adult patients in the age group of 20-60 years belonging to ASA I and II scheduled to undergo elective upperlimb orthopaedic procedures were chosen. All the patients were assessed and those with normal clinical, hematological, biochemical and radiological parameters were selected.

Informed written consent was obtained from all the patients. All the patients were randomnly assigned to two groups Group-A and Group-B each containing 30 patients.

Group-A: Patients undergoing supraclavicular brachial plexus block by ultrasound guided technique with 25ml of 0.25% Bupivacaine.

Group-B: Patients undergoing supraclavicular brachial plexus block by ultrasound guided technique with 25ml of 0.25% Bupivacaine and 3μg/Kg of Buprenorphine.

INCLUSION CRITERIA

- Assessed patients of ASA I and II status.
- Both gender.
- Normal biochemical and hematological parameters.
- Age group between 20 -60years
- Patients weighing 50-70 Kgs.
- Mallampati score of I and II
- No known neurological deficit
- No local sepsis
- Those who have given valid informed consent

EXCLUSION CRITERIA

- Patients who have not given consent.
- Patients with anticipated difficult airway.
- Neurological disorders

- History of allergy to local anaesthetics
- History of cardiovascular and respiratory disorders
- Local bony deformities
- History of bleeding disorders
- Extremely obese patients

MATERIALS REQUIRED

- Equipments for the block
- Ultrasound machine
- Sterile tray for regional blocks
- Drugs for the block- 0.25% Bupivacaine, Buprenorphine
- Equipment and drugs for resuscitation
- Equipments and drugs for conversion to general anaesthesia in case of block failure

METHODS

PREOPERATIVE PREPARATION

Patients were assessed preoperatively and the procedure was explained to the patients. Written informed consent was obtained.

Assessment of pain using VRS- Verbal Rating Scale intraoperatively and VAS- Visual analogue score post operatively was explained to the patients preoperatively.

PREMEDICATION

Patients were not given any premedication.

CONDUCT OF ANAESTHESIA

On arrival of the patient in the operating room, monitors were connected. Monitors include pulse oximetry, NIBP and ECG and baseline vitals signs were recorded. An intravenous access was obtained in the opposite arm. The patients were given ultrasound guided supraclavicular brachial plexus block as follows.

PROCEDURE

The patient is made to lie supine with a small pillow below head and neck and turned the head to the side opposite to that to be blocked.

The USG probe is cleaned and covered in a sterile cover and the patients' skin is prepared with povidone iodine and drapped with sterile towel. The probe is placed and adjusted to visualize the brachial plexus and local anaesthetic solution is injected onto the skin at the needle entry point.

A 100mm long needle is inserted 1 to 2cm away from the central border of the probe. The needle angle is maintained at 0-45˚.

"Inplane" approach is used for the block. Needle is inserted initially in the superficial plane until the needle is visualized on the scan. Once the hyperechoic line is seen, it is inserted towards the brachial plexus and placed in the cornerpocket. Throughout the procedure the needle tip was traced. After careful negative aspiration, local anaesthetic 1-2ml was injected to see the spread thereby confirming the needle tip position. Then, by adjusting the needle position, drug is deposited all around the brachial plexus.

EVALUATION OF THE BLOCK

The following observations were made

1) Vital signs monitoring: Non- invasive blood pressure and heart rate was measured every 1 minute for the first 10 minutes and every 5 minutes thereafter throughout the intra operative period. ECG and SPO2, respiratory rate were monitored continuously. For statistical purposes, they were documented at 1, 5, 10, 15, 30 minutes and every 30 minutes till 2 hours and every 2 hours thereafter.

2) Immediately following the administration of the drug, patients were evaluated every minute till the onset of sensory and motor blockade.

3) Time of onset of sensory blockade was tested by assessment of pinprick sensation with a 26G hollow needle.

4) Onset of motor blockade was assessed by loss of shoulder abduction. Failure of the block to appear in 20 minutes was taken as failure and

the patients were administered general anaesthesia and were excluded from the study.

5) After confirmation that the block has taken up, surgery was started. During the surgical procedure, the degree of pain was assessed with a 3 point verbal rating score (VRS).

0- No Pain

1- Pain

2- Unbearable Pain

If VRS >1, patients were administered general anaesthesia and were excluded from the study.

6) Local anaesthetic toxicity manifestations like circumoral numbness, tinnitus, twitching, convulsions etc, were looked for and appropriate measures were planned.

7) Complications like intravascular injection and pneumothorax were looked for and appropriate measures were planned.

8) Duration of analgesia were tested postoperatively using VAS score every ½ Hour for the first 6 hours thereafter every 1 hour till 12 hours, thereafter every 4 hours till 24 hours.

Patients were given rescue analgesia with intramuscular diclofenac 75 mg when VAS > 4.

9) Side Effects of opioids are to be looked for 48 Hours.

a. Nausea and vomiting

b. Pruritis

c. Urinary Retention

d. Hypotention

e. Headache

f. Respiratory Depression defined as RR <10/ minute

g. Any other neurological depression

PARAMETERS STUDIED

Onset of Sensory Analgesia

This is the time in minutes from the injection of the drug to the lack of appreciation of pinprick sensation.

Onset of motor blockade

This is the time in minutes from the time of drug injection to the loss of shoulder abduction.

Duration of Analgesia

This is the time in hours from the onset of analgesia to the time of administration of rescue analgesia.

Side effects of Buprenorphine

All the patients included in the study were monitored for a period of 24 hours from the time of administration the drug. All the parameters were subjected to statistical analysis.

STATISTICAL ANALYSIS

The data were analysis using SPSS (Statistical Package for Social Science) Version 16.01. The data collected were scored and analyzed, Continuous variables were presented as means with Standard deviation (sd) and categorical variables were presented as frequency and percentages. Student t-test was used for testing the significance of all the variables (Mean & Sd) in both the group. Chi-square test was used to compare proportions. All the Statistical results were considered significant at P value = 0.05.

Sample Selection

Pilot study was done with a sample size of 6 patients in each group, before the start of the study to decide on sample size. The mean and standard deviation of insertion time was calculated from pilot study. The sample size is calculated based on the formula given in NTI Bulletin 2006. (Sample size determination in health studies, V.K.Chadha,Sr. Epidemiologist, National Institute Bulletin 2006, 42/3 & 4m 55-62.).

From the pilot study the pilot study got the value of mean and standard deviation of duration of Analgesia of Group-1 (4.43 ± 0.62) and Group-II (5.05 ± 1.65).

$$n = \frac{[Z_{1-\alpha/2} + Z_{1-\beta}]^2 (2\sigma^2)}{(d)} = (8.98 * 5.09)/ 0.38$$

n = 23.132/0.384

n =60.24 (60)

$Z_1 - \alpha/2 = 1.96$ (5%)

$Z_{1-\beta}$ = 1.037 (85 % Power)

$[Z_{1-\alpha/2} + Z_{1-\beta}]^2 = (1.96 + 1.037)^2 = 8.98$

$S = (s_1 + s_2) / 2$

S =(0.62+1.65)/2 =2.27/2 =1.135

$S^2 = (1.135)^2 = 1.288$

$2\sigma^2 = 1.288 * 2 = 2.57645$ 2.576

d=(Mean1 –Mean2)

= (4.43-5.05)= -0.62

$d^2 = 0.3844$

From the above calculation sample size was decided as 60 (30 for each group)

OBSERVATIONS AND RESULTS

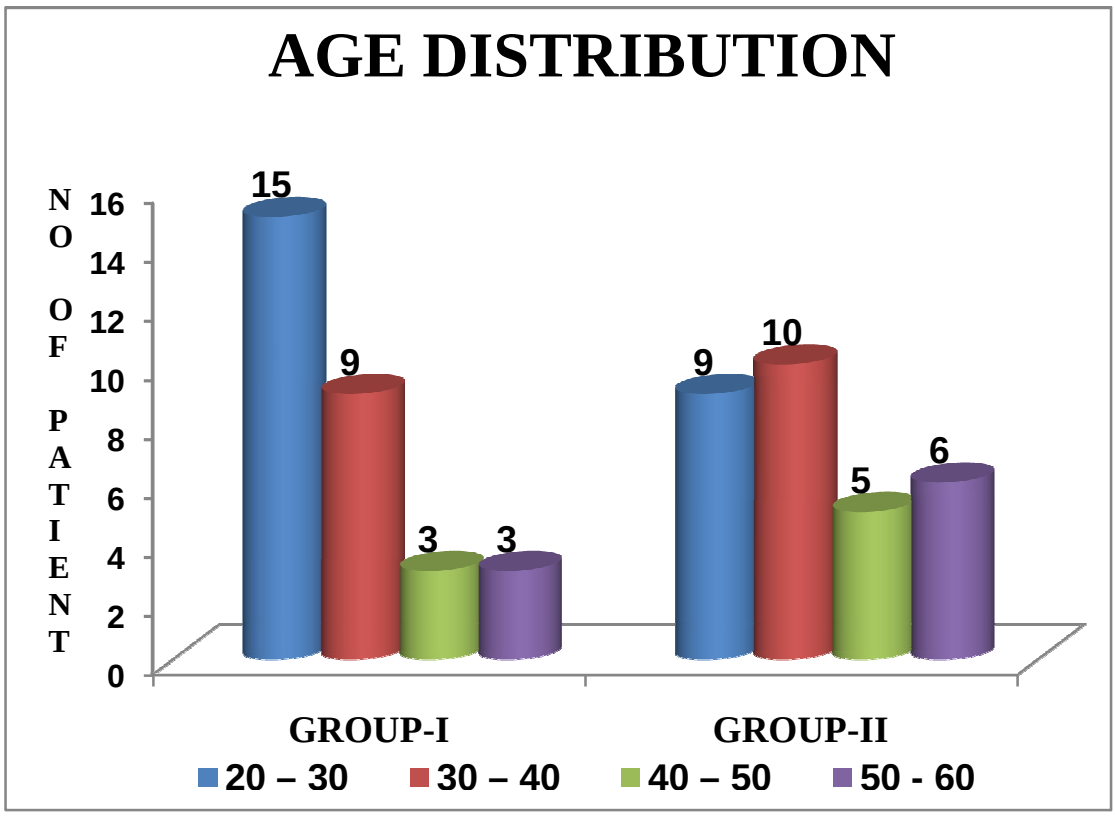
The patients included in this study were divided into two groups consisting of 30 patients each.

Group I (n =30) received 0.25% bupivacaine

Group II (n =30) received 0.25% bupivacaine + 3 microgram/kg buprenorphine.

AGE

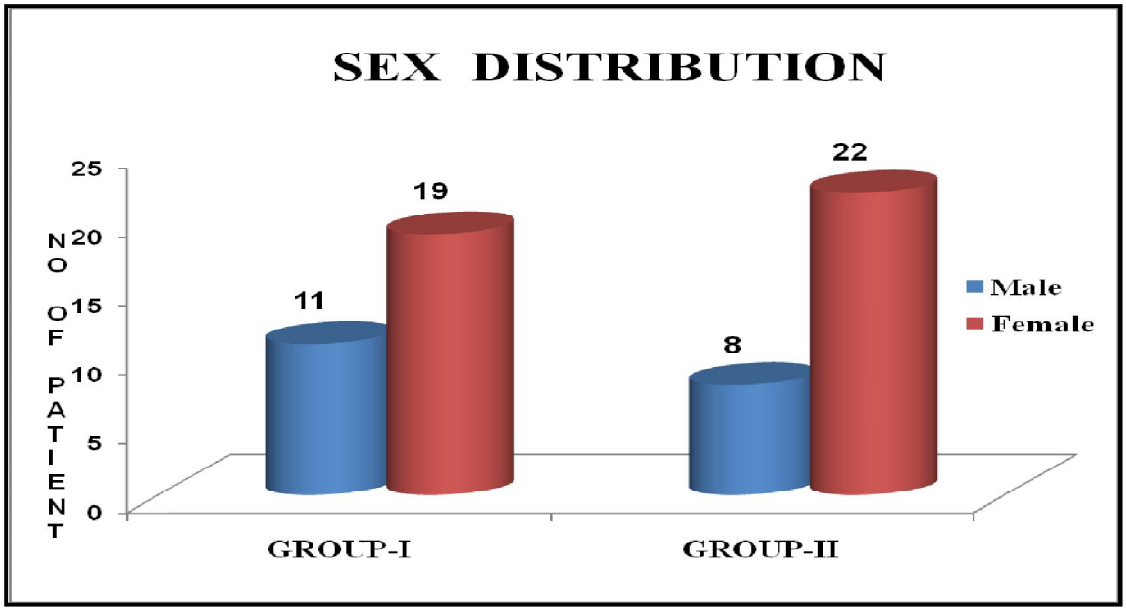
Age Group	GROUP-I		GROUP-II	
	No of Patients(N)	Percentage (%)	No of Patients(N)	Percentage (%)
20 – 30	15	50.00	9	30.00
30 – 40	9	30.00	10	33.30
40 – 50	3	10.00	5	16.70
50 – 60	3	10.00	6	20.00
TOTAL	30	100	30	100
Mean	33.37		38.17	
Sd	11.17		11.11	
t- value	1.67			
p-value	0.10			
Significance	Not Significant			



Majority of the Group I patients belonged to 20 -30 years age group (n =15, 50%) with a mean age group of 33.37 years. In Group II patients, majority belonged to 30 -4- years age group(n =10, 33.3%) with a mean age group of 38.17 years. The association between the intervention groups and age distribution is considered to be not statistically significant since p> o.o5 as per student t-test.

SEX

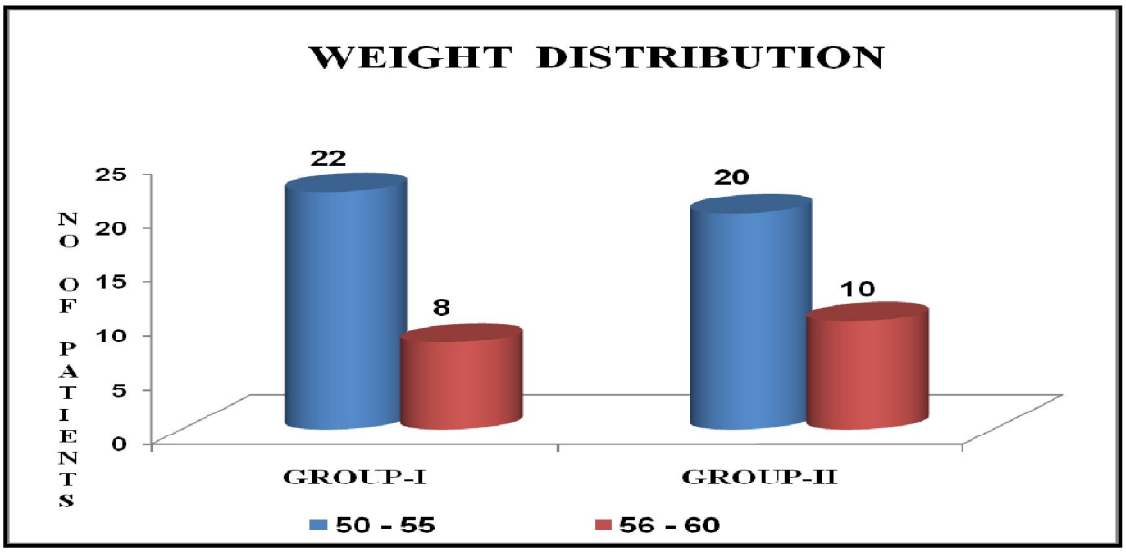
Sex	GROUP-I		GROUP-II	
	No of Patients(N)	Percentage (%)	No of Patients (N)	Percentage (%)
Male	11	36.67	8	26.67
Female	19	63.33	22	73.33
Total	30	100	30	100
Ratio(Male : Female)	11 : 19		8 : 22	
Chi-Square	0.69			
p-value	0.41			
Significance	Not Significant			



Majority of the Group I patients belonged to female gender (n= 19,63.33%). In Group II patients also, majority belonged to female gender (n =22,73.33%).The association between the intervention groups and gender distribution is considered to be not statistically significant since p >0.05 as per chi square test.

WEIGHT

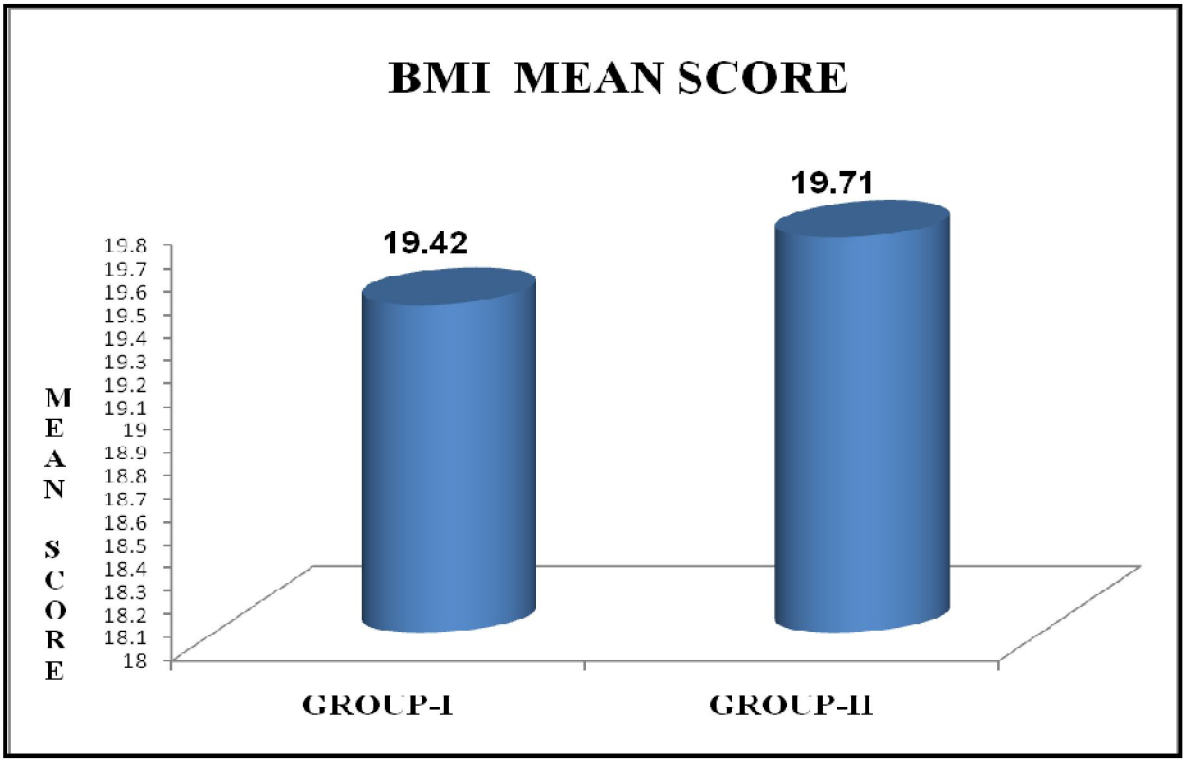
Weight	GROUP-I		GROUP-II	
	No of Patients(N)	Percentage (%)	No of Patients(N)	Percentage (%)
50 – 55	22	73.33	20	66.67
56- 60	8	26.67	10	33.33
TOTAL	30	100	30	100
Mean	57.37		58.40	
Sd	3.74		3.64	
t- value	1.09			
p-value	0.28			
Significance	Not Significant			



Majority of the Group I patients belonged to 50 -55 kgs (n =22, 73.33%) with a mean weight of 57.37 kgs. In Group II patients also, majority belonged to 50-55 kgs (n =20,66.67%) with a mean weight of 58.40 kgs. The association between intervention groups and weight distribution is considered to be not statistically significant since p> 0.05 as per student t- test.

BODY MASS INDEX

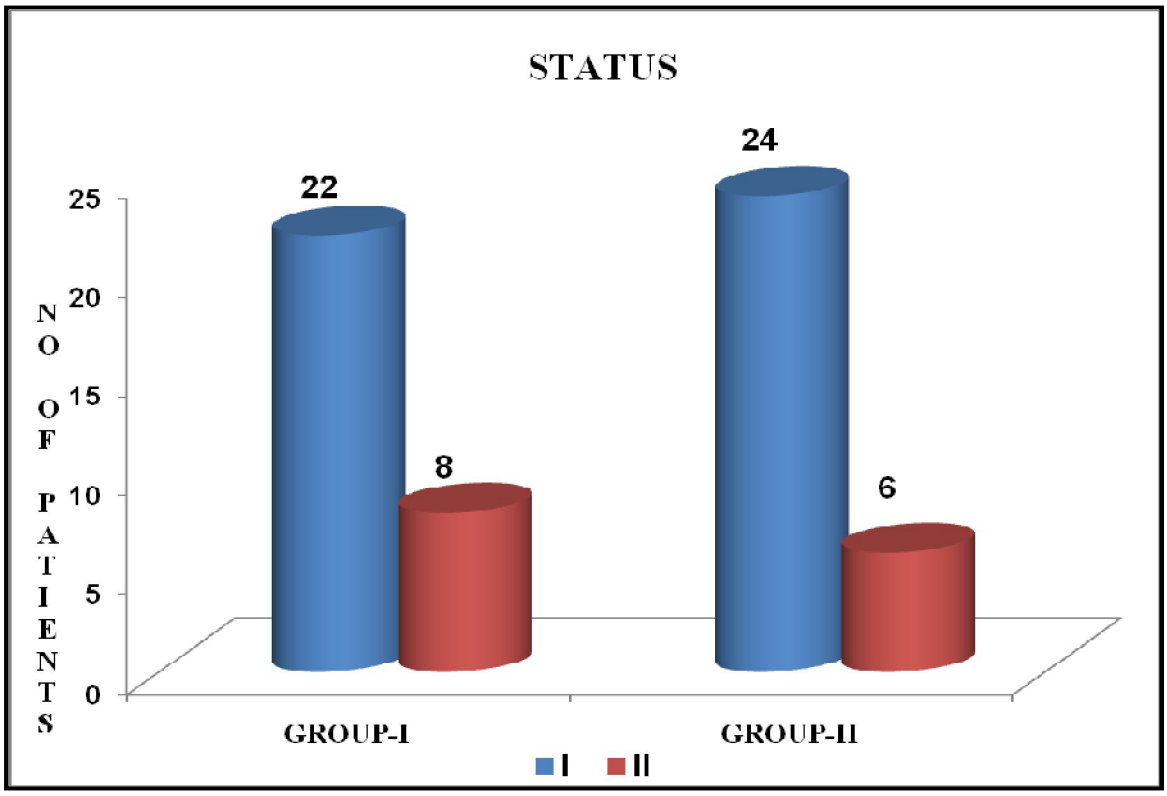
Group	Mean	Standard Deviation
GROUP-I	19.42	0.97
GROUP-II	19.71	0.89
t-value	1.23	
p-value	0.22	
Significance	**Not Significant**	



In Group I patients the mean body mass index is 19.42 with standard deviation of 0.97. In Group II patients, the mean body mass index is 19.71 with standard deviation of 0.89. The association between the intervention groups and body mass index distribution is considered to be not statistically significant since $p > 0.05$ as per student t- test.

ASA STATUS

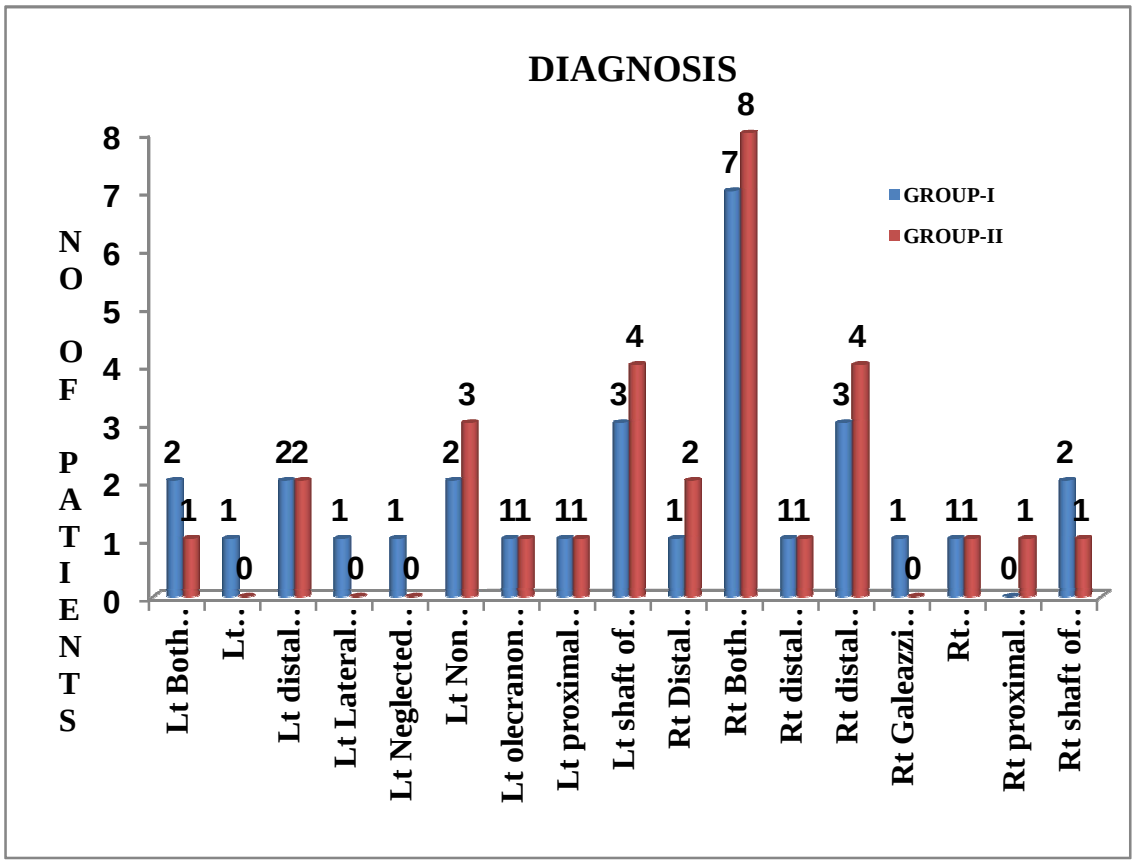
STATUS	GROUP-I		GROUP-II	
	No of Patients(N)	Percentage (%)	No of Patients(N)	Percentage (%)
I	22	73.33	24	80
II	8	26.67	6	20
TOTAL	30	100	30	100



Majority of Group I patients belonged to ASA I (n = 22, 73.33%). In Group II patients also, majority belonged to ASA I (n = 24, 80%)

DIAGNOSIS

DIAGNOSIS	GROUP-I		GROUP-II	
	No of Patients (N)	Percentage (%)	No of Patients (N)	Percentage (%)
Lt Both Bone fore arm Fracture	2	6.67	1	3.33
Lt Capitulum Fracture	1	3.33	0	0
Lt distal radius Fracture	2	6.67	2	6.67
Lt Lateral Condyle humerus	1	3.33	0	0
Lt Neglected Elbow dislocation	1	3.33	0	0
Lt Non Union radius	2	6.67	3	10
Lt olecranon fracture	1	3.33	1	3.33
Lt proximal Ulna Fracture	1	3.33	1	3.33
Lt shaft of humerus fracture	3	10.00	4	13.33
Rt Distal humerus fracture	1	3.33	2	6.67
Rt Both bone forearm fracture	7	23.33	8	26.68
Rt distal humerus fracture	1	3.33	1	3.33
Rt distal radius fracture	3	10.00	4	13.34
RtGaleazzi fracture	1	3.33	0	0
Rt olecranon fracture	1	3.33	1	3.33
Rt proximal ulna fracture	0	0.00	1	3.33
Rt shaft of humerus fracture	2	6.67	1	3.33
TOTAL	30	100	30	100

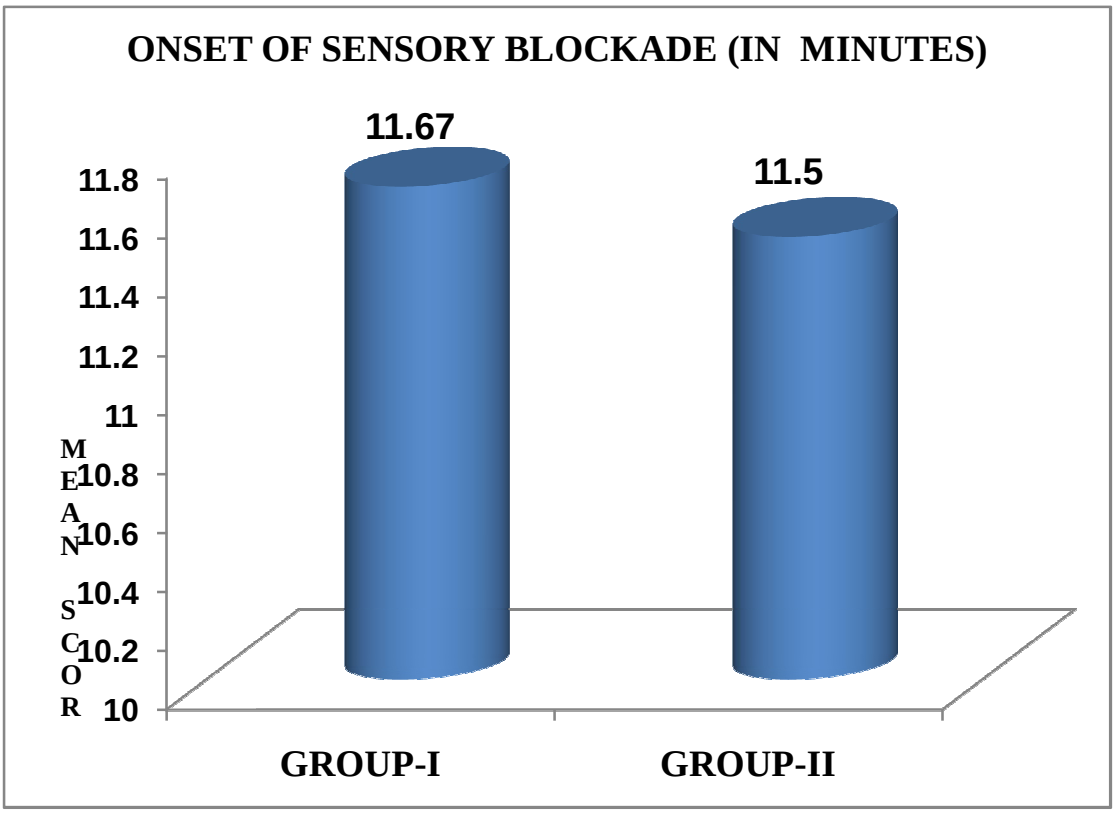


Majority of Group I patients had both bone fracture (n =7, 23.33%).

In Group II patients also, majority had both bone fracture(n =8, 26.68%).

ONSET OF SENSORY BLOCKADE (IN MINUTES)

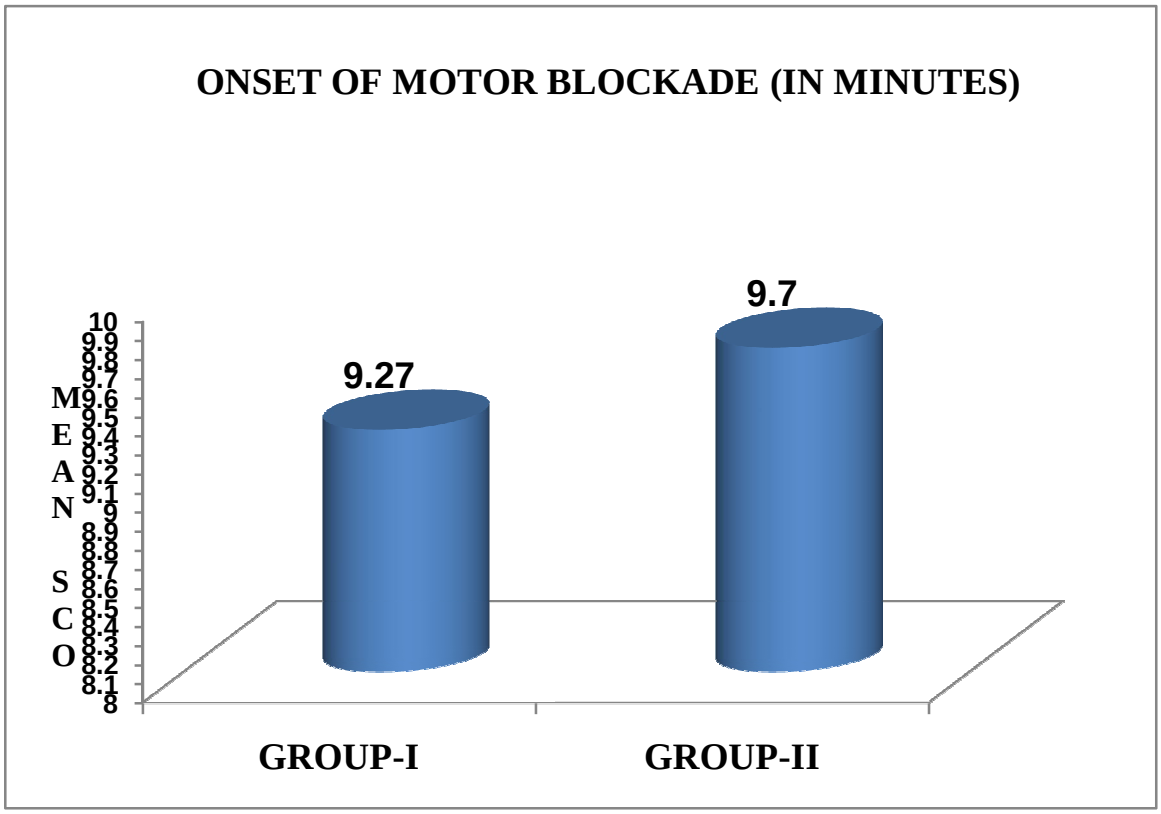
Group	Mean	Standard Deviation
GROUP-I	11.67	0.76
GROUP-II	11.50	0.90
t-value	0.78	
p-value	0.44	
Significance	Not Significant	



The mean time of onset of sensory blockade in Group I is 11.67 minutes with standard deviation of 0.76. The mean onset time in Group II is 11.50 minutes with standard deviation of 0.90. The association between the intervention groups and onset of sensory blockade is considered to be not statistically significant since $p > 0.05$ as per student t- test.

ONSET OF MOTOR BLOCKADE (IN MINUTES)

Group	Mean	Standard Deviation
GROUP-I	9.27	0.74
GROUP-II	9.70	0.79
t-value	2.19	
p-value	0.03	
Significance	Significant	



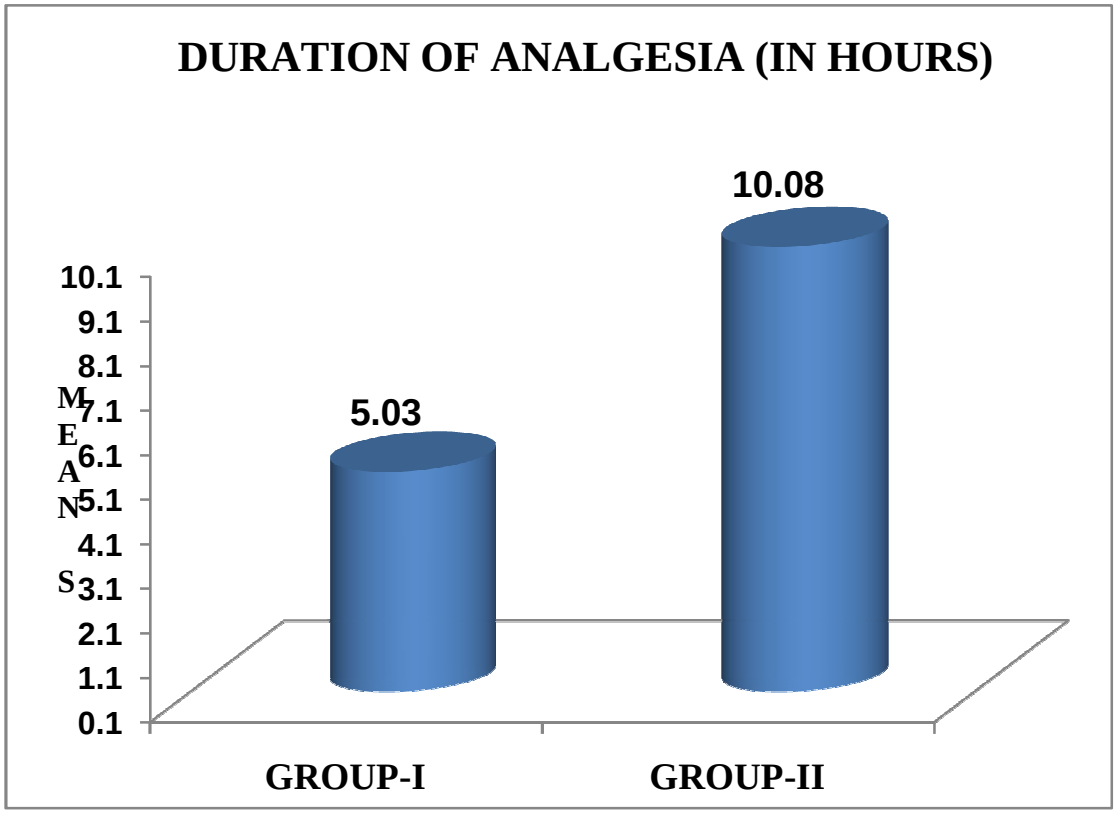
The mean time of onset of motor blockade in Group I patients is 9.27 minutes with standard deviation of 0.74. The mean time of onset in Group II patients is 9.70 minutes with standard deviation of 0.79. The association between intervention groups and onset of motor blockade is considered to be statistically significant since p <0.05 indicating true difference between intervention groups.

CLINICAL SIGNIFICANCE:

The mean time of onset of motor blockade was meaningfully less in bupivacaine group compared to buprenorphine group by 0.43 minutes with a p value of 0.03 as per student t- test. This significant difference in decrease in onset of motor blockade in bupivacaine group compared to buprenorphine group is true and has not occurred by chance.

DURATION OF ANALGESIA (IN HOURS)

Group	Mean	Standard Deviation
GROUP-I	5.03	0.24
GROUP-II	10.08	0.44
t-value	55.00	
p-value	0.0001	
Significance	**Significant**	



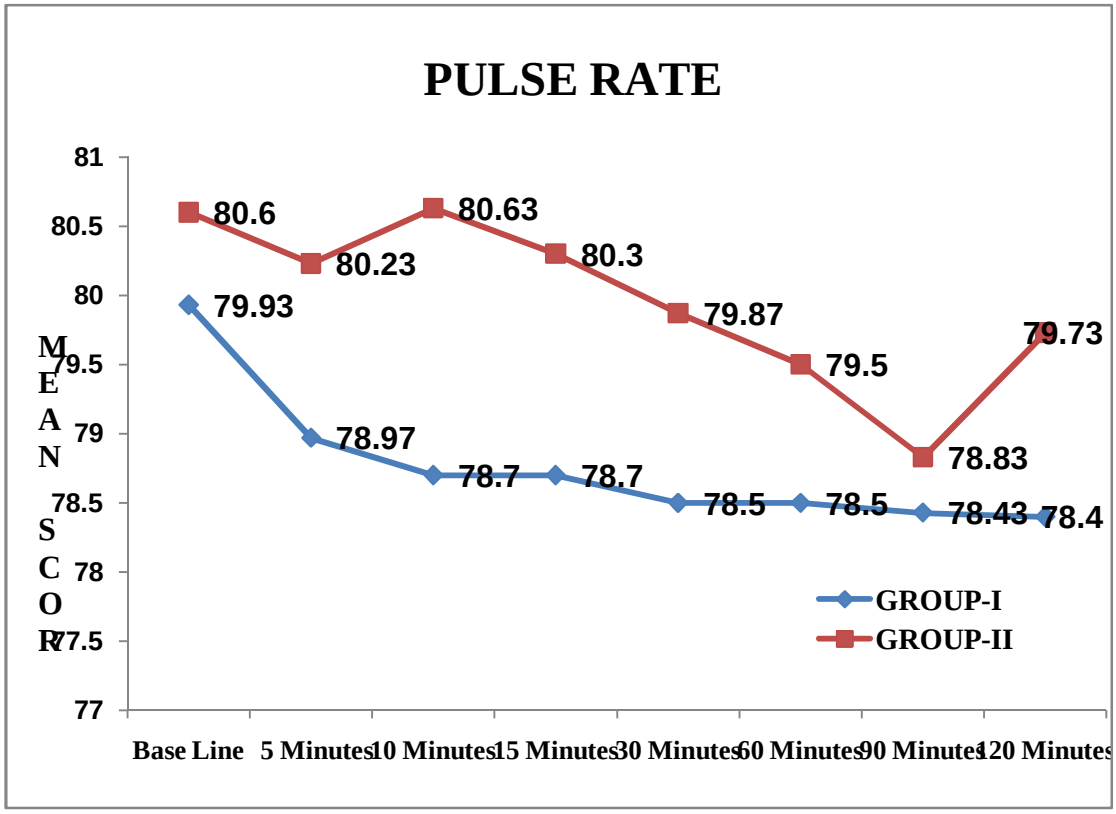
The mean duration of analgesia in Group I is 5.03 hours with standard deviation of 0.24.The mean duration of analgesia in Group II is 10.08 hours with standard deviation of 0.44. The association between intervention groups and duration of analgesia is considered to be statistically significant since $p < 0.05$ as per student t- test indicating true difference among intervention groups.

CLINICAL SIGNIFANCE:

The mean duration of analgesia was meaningfully increased in buprenorphine group as compared to bupivacaine group by 5.05 hours with a p value of 0.0001 as per student t- test. This significant difference in increase in duration of analgesia in buprenorphine group compared to bupivacaine group is true and not occurred by chance and we can safely conclude that buprenorphine added to bupivacaine in supraclavicular brachial plexus block in patients undergoing elective upper limb surgeries significantly increase the duration of analgesia compared to bupivacaine alone.

PULSE RATE

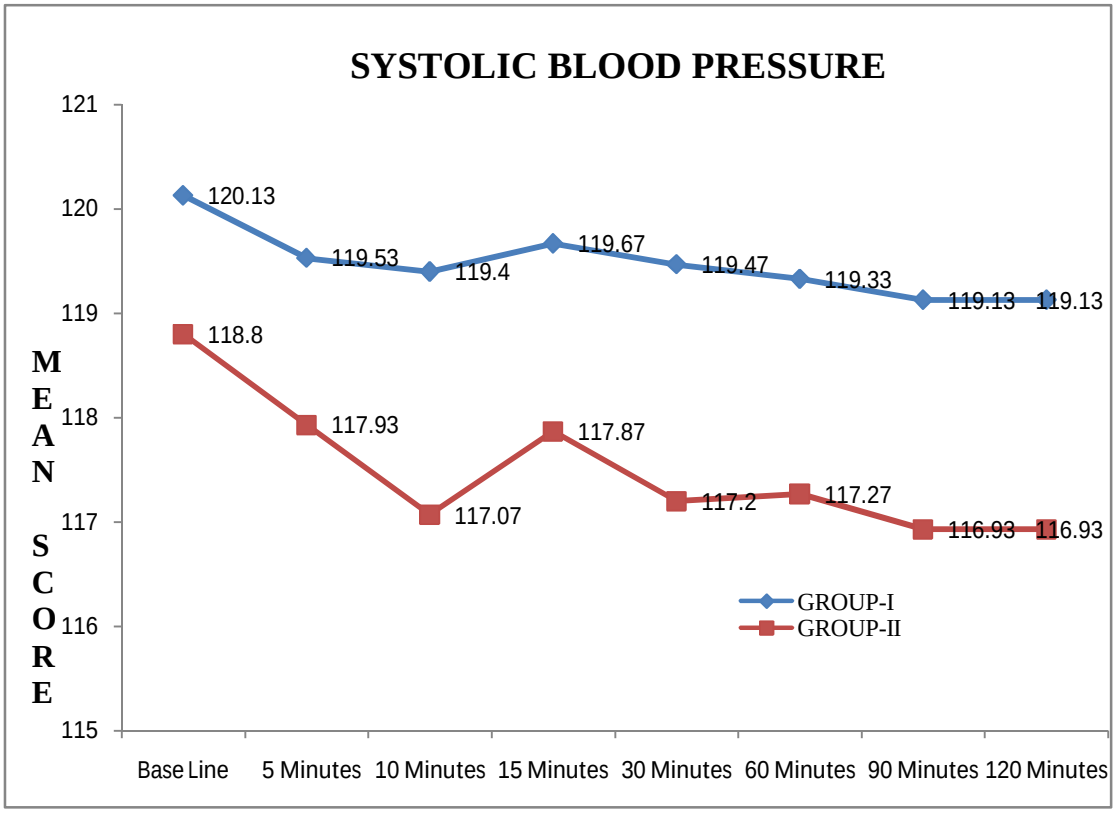
Time	GROUP-I		GROUP-II		t-value	p-value	Significance
	Mean	Sd	Mean	Sd			
Base Line	79.93	12.02	80.60	10.87	0.23	0.82	**NS**
5 Minutes	78.97	12.01	80.23	9.87	0.45	0.66	**NS**
10 Minutes	78.70	10.76	80.63	9.72	0.73	0.47	**NS**
15 Minutes	78.70	10.51	80.30	8.57	0.65	0.52	**NS**
30 Minutes	78.50	11.02	79.87	9.79	0.51	0.61	**NS**
60 Minutes	78.50	10.44	79.50	9.44	0.39	0.70	**NS**
90 Minutes	78.43	10.69	78.83	9.82	0.15	0.88	**NS**
120 Minutes	78.40	10.02	79.73	9.77	0.52	0.60	**NS**



Most of the bupivacaine group patients had mean pulse rate ranging from 78.40 bpm to 79.93 bpm between 0 minutes and 120 minutes. Similarly bupreorphine group patients had mean pulse rate ranging from 78.83 bpm to 80.63 bpm between 0 minutes and 120 minutes. The association between the intervention groups and pulae rate is considered to be not statistically significant since p >0.05 as per student t- test.

SYSTOLIC BLOOD PRESSURE

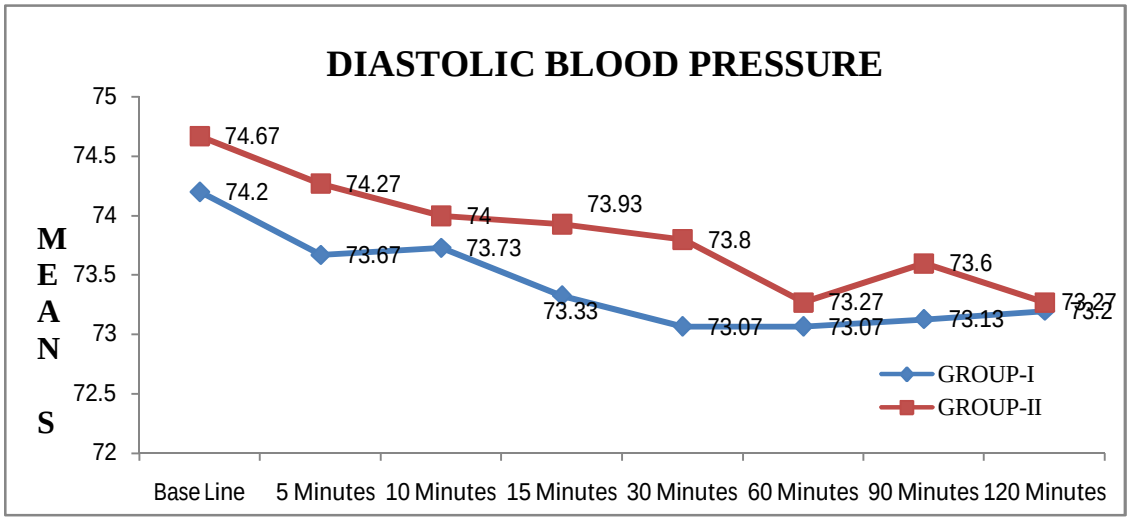
Time	GROUP-I		GROUP-II		t-value	p – value	Significance
	Mean	Sd	Mean	Sd			
BaseLine	120.13	6.81	118.80	6.21	0.79	0.43	NS
5Minutes	119.53	7.91	117.93	7.31	0.81	0.42	NS
10Minutes	119.40	6.95	117.07	7.25	1.27	0.21	NS
15Minutes	119.67	6.19	117.87	5.63	1.83	0.07	NS
30Minutes	119.47	7.03	117.20	5.16	1.42	0.16	NS
60Minutes	119.33	6.35	117.27	3.98	1.51	0.14	NS
90Minutes	119.13	4.66	116.93	5.63	1.65	0.10	NS
120Minutes	119.13	5.50	116.93	4.60	1.68	0.10	NS



Most of the bupivacaine group patients had mean systolic blood pressure ranging from 119.13 mmHg to 120.13 mmHg between 0 minutes and 120 minutes. Similarly in buprenorphine group patients, the mean blood pressure ranging from 116.93 mmHg to 118.80 mmHg between 0 minutes and 120 minutes. The association between the intervention groups and systolic blood pressure is considered to be not statistically significant since $p > 0.05$ as per student t-test.

DIASTOLIC BLOOD PRESSURE

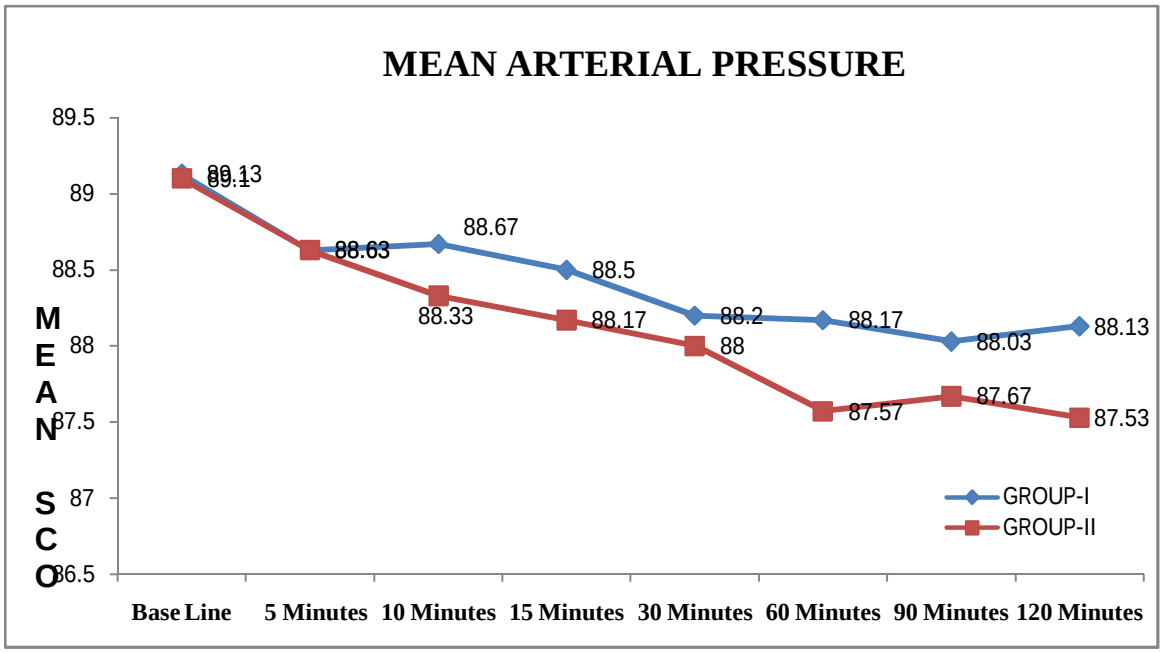
Time	Group-I		Group-II		t-value	p-value	Significance
	Mean	Sd	Mean	Sd			
BaseLine	74.20	4.62	74.67	3.25	0.45	0.65	NS
5Minutes	73.67	5.10	74.27	3.74	0.52	0.61	NS
10Minutes	73.73	5.84	74.00	3.32	0.22	0.83	NS
15 Minutes	73.33	6.16	73.93	2.95	0.48	0.63	NS
30 Minutes	73.07	4.84	73.80	2.43	0.74	0.46	NS
60 Minutes	73.07	4.54	73.27	1.34	0.23	0.82	NS
90 Minutes	73.13	4.19	73.60	1.43	0.58	0.57	NS
120 Minutes	73.20	4.35	73.27	2.00	0.08	0.94	NS



Most of the bupivacaine group patients had mean diastolic blood pressure ranging from 73.07 mmHg to 74.20 mmHg between 0 minutes and 120 minutes. Similarly in buprenorphine group patients, the mean diastolic blood pressure was ranging from 73.27 mmHg to 74.0 mmHg between 0 minutes and 120 minutes. The association between the intervention groups and diastolic blood pressure is considered to be not statistically significant since p >0.05 as per student t-test.

MEAN ARTERIAL PRESSURE

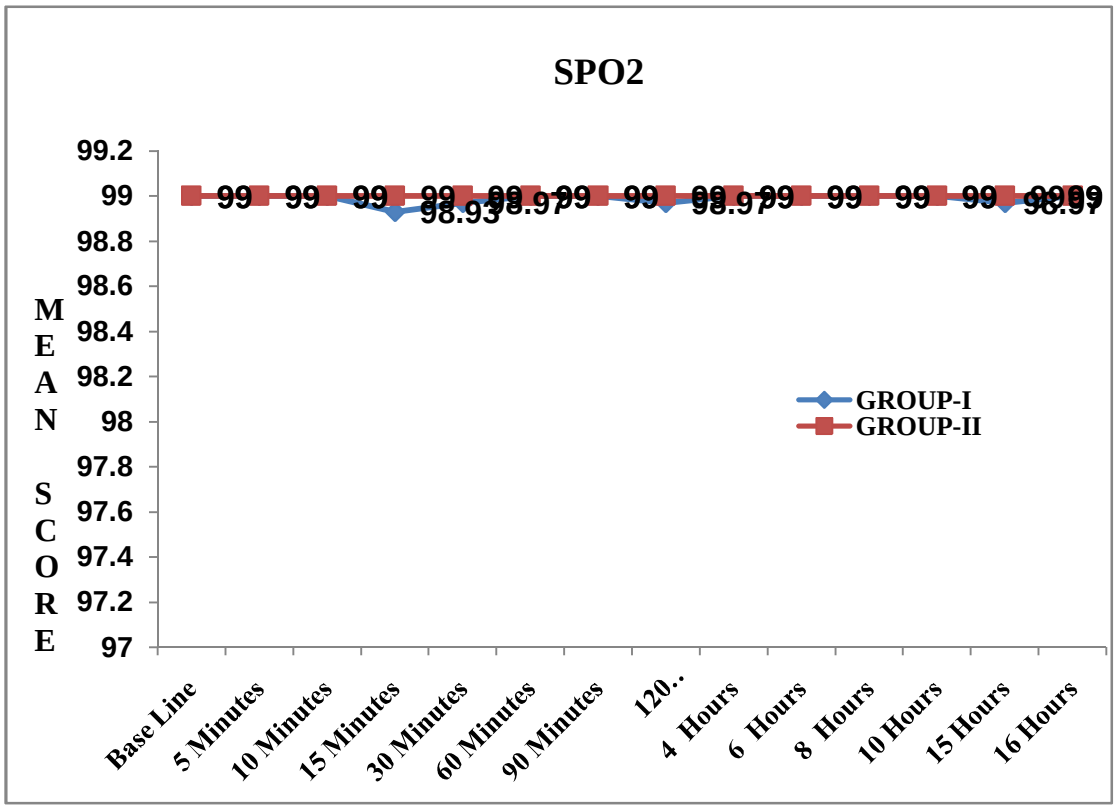
Time	GROUP-I		GROUP-II		t-value	p-value	Significance
	Mean	Sd	Mean	Sd			
BaseLine	89.13	4.90	89.10	3.45	0.03	0.98	NS
5Minutes	88.63	5.68	88.63	4.04	0.01	1.00	NS
10Minutes	88.67	5.34	88.33	3.33	0.29	0.77	NS
15Minutes	88.50	5.23	88.17	2.71	0.31	0.76	NS
30Minutes	88.20	4.58	88.00	2.48	0.21	0.83	NS
60Minutes	88.17	4.87	87.57	1.81	0.63	0.53	NS
90Minutes	88.03	4.05	87.67	2.17	0.44	0.66	NS
120Minutes	88.13	4.21	87.53	2.08	0.70	0.49	NS



Most of the bupivacaine group patients had mean arterial pressure ranging from 88.03 mmHg to 89.13 mmHg betweeen 0 minutes and 120 minutes. Similarly the buprenorphine group patients had mean arterial pressure ranging from 87.53 mmHg to 89.10 mmHg between 0 minutes and 120 minutes. The association between the intervention groups and the mean arterial pressure is onsidered to be not statistically significant since p >0.05 as per student t-test.

SpO2

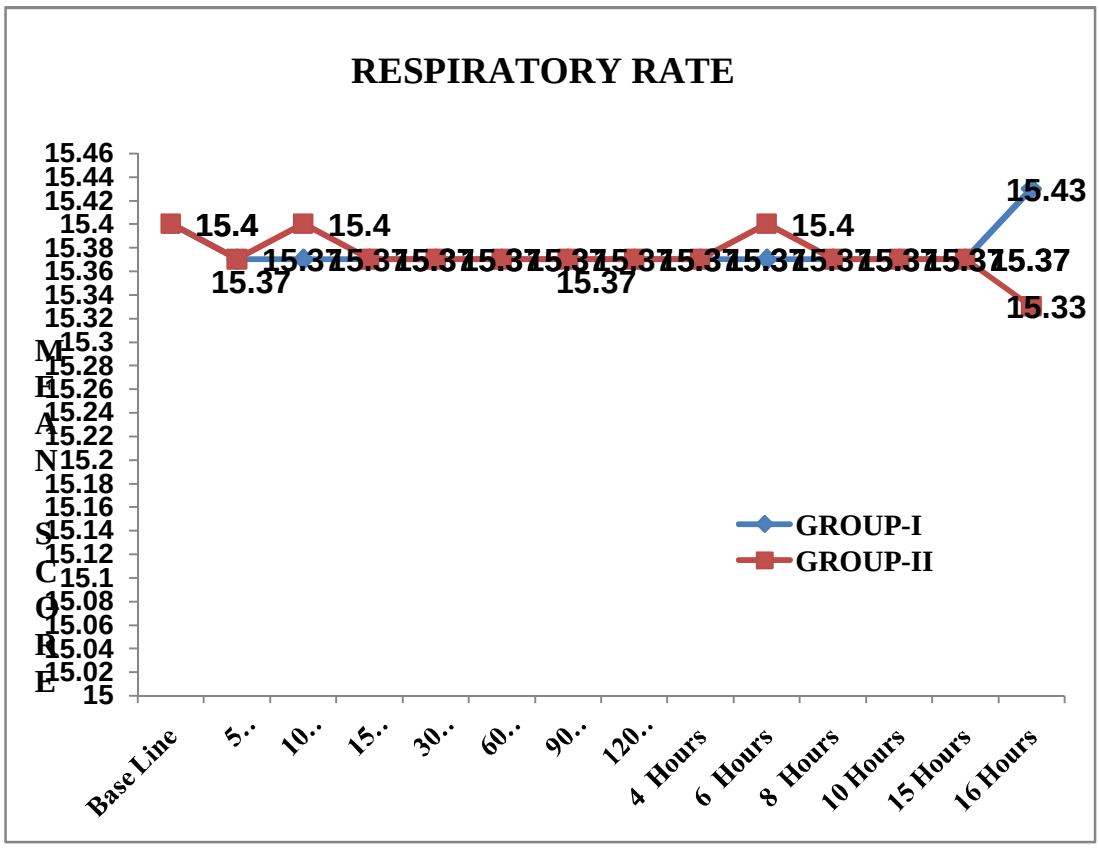
Time	GROUP-I		GROUP-II		t-value	p-value	Significance
	Mean	Sd	Mean	Sd			
BaseLine	99.00	0.00	99.00	0.00	-	-	-
5Minutes	99.00	0.00	99.00	0.00	-	-	-
10Minutes	99.00	0.00	99.00	0.00	-	-	-
15Minutes	98.93	0.25	99.00	0.00	1.44	0.16	NS
30Minutes	98.97	0.18	99.00	0.00	1.00	0.32	NS
60Minutes	99.00	0.00	99.00	0.00	-	-	-
90Minutes	99.00	0.00	99.00	0.00	-	-	-
120Minutes	98.97	0.18	99.00	0.00	1.00	0.32	NS
4 Hours	99.00	0.00	99.00	0.00	-	-	-
6 Hours	99.00	0.00	99.00	0.00	-	-	-
8 Hours	99.00	0.00	99.00	0.00	-	-	-
10 Hours	99.00	0.00	99.00	0.00	-	-	-
12 Hours	98.97	0.18	99.00	0.00	1.00	0.32	NS
16 Hours	99.00	0.00	99.00	0.00	-	-	-



Most of the bupivacaine group patients had mean spo2 ranging from 98.97% to 99% between 0 minutes and 16 hours. Similarly in buprenorphine group patients the mean spo2 was 99% between 0 minutes and 16 hours. The association between the intervention groups and spo2 is considered to be not statistically significant since $p > 0.05$ as per student t- test.

RESPIRATORY RATE

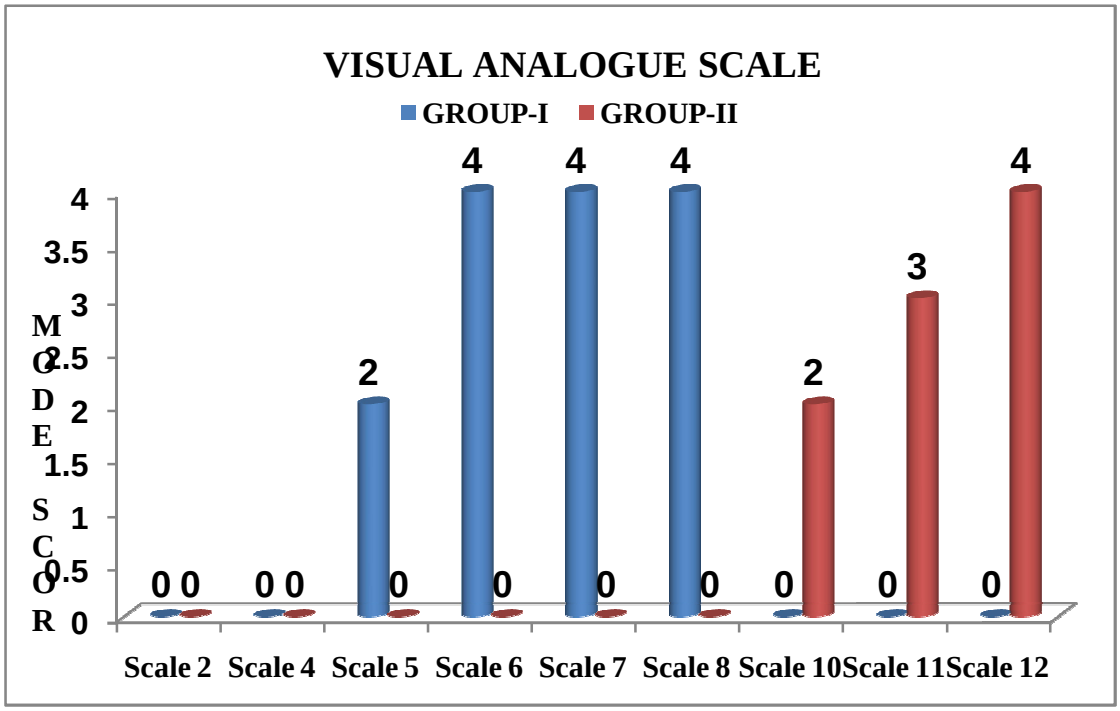
Time	GROUP-I		GROUP-II		t-value	p-value	Significance
	Mean	Sd	Mean	Sd			
BaseLine	15.40	0.50	15.40	0.50	0.01	1.00	**NS**
5Minutes	15.37	0.49	15.37	0.49	0.01	1.00	**NS**
10Minutes	15.37	0.49	15.40	0.50	0.26	0.80	**NS**
15Minutes	15.37	0.49	15.37	0.49	0.01	1.00	**NS**
30Minutes	15.37	0.49	15.37	0.49	0.26	0.80	**NS**
60Minutes	15.37	0.50	15.37	0.50	0.01	1.00	**NS**
90Minutes	15.37	0.50	15.37	0.49	0.01	1.00	**NS**
120Minutes	15.37	0.49	15.37	0.49	0.01	1.00	**NS**
4 Hours	15.37	0.49	15.37	0.49	0.01	1.00	**NS**
6 Hours	15.37	0.49	15.40	0.50	0.26	1.00	**NS**
8 Hours	15.37	0.49	15.37	0.49	0.01	1.00	**NS**
10 Hours	15.37	0.49	15.37	0.49	0.01	1.00	**NS**
12 Hours	15.37	0.62	15.37	0.49	0.01	1.00	**NS**
16 Hours	15.43	0.57	15.33	0.48	0.74	0.46	**NS**



Most of the bupivacaine group patients had respiratory rate ranging from 15.37 breaths/min to 15.43 breaths/min between 0 minutes and 16 hours. Similarly in buprenorphine group patients, the respiratory rate ranging from 15.33 breaths/min to 15.40 breaths/min between 0 minutes and 16 hours. The association between the intervention groups and respiratory rate is considered not to be statistically significant since p >0.05 as per student t- test.

VISUAL ANALOGUE SCALE

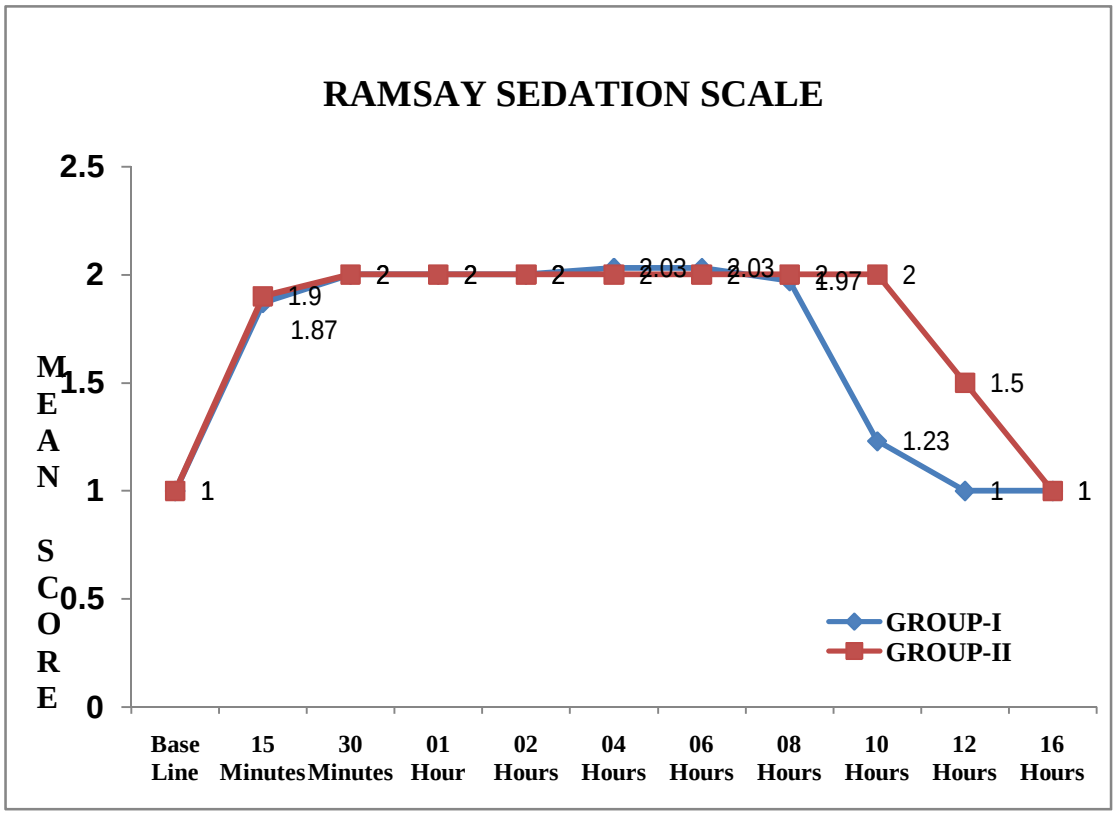
Time	Group-I	Group-II
	Mode	Mode
Scale 2	0	0
Scale 4	0	0
Scale 5	2	0
Scale 6	4	0
Scale 7	4	0
Scale 8	4	0
Scale 10	0	2
Scale 11	0	3
Scale 12	0	4



Most of the bupivacaine group patients had VAS score >4 at 6 hours, 7 hours and 8 hours postoperatively while in buprenorphine group, most of the pateints had VAS > 4 at 12 hours postoperatively. The association between intervention groups and VAS is considered to be statistically significant.

RAMSAY SEDATION SCALE

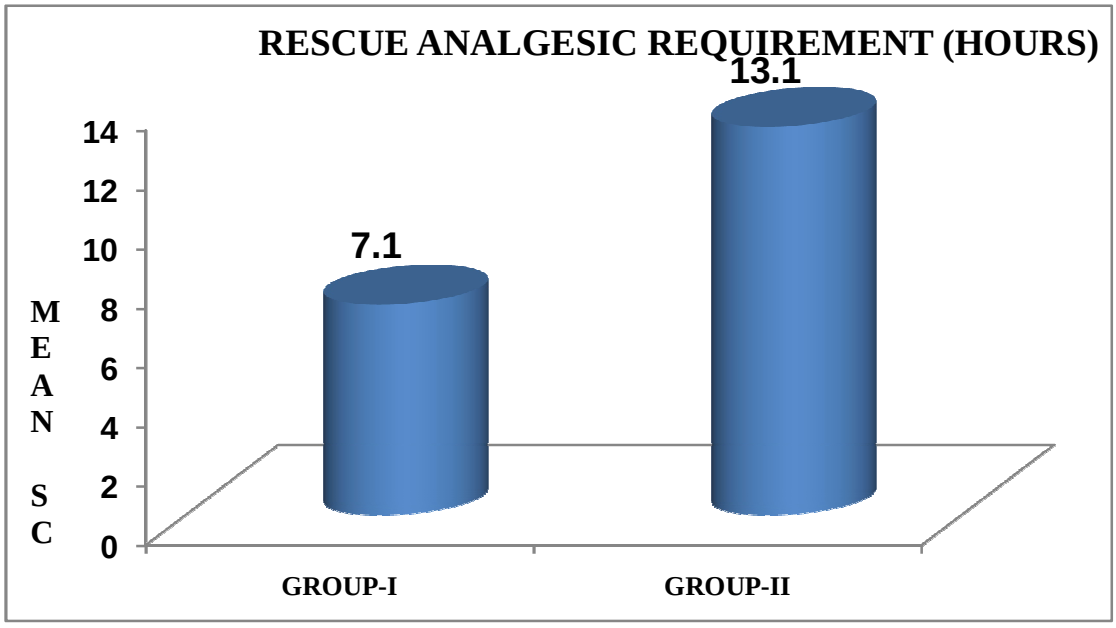
Time	GROUP-I		GROUP-II		t-value	p-value	Significance
	Mean	Sd	Mean	Sd			
BaseLine	1.00	0.00	1.00	0.00	-	-	NS
15Minutes	1.87	0.35	1.90	0.31	0.40	0.69	NS
30Minutes	2.00	0.00	2.00	0.00	-	-	NS
1 Hour	2.00	0.00	2.00	0.00	-	-	NS
2 Hours	2.00	0.00	2.00	0.00	-	-	NS
4 Hours	2.03	0.18	2.00	0.00	1.00	0.32	NS
6 Hours	2.03	0.18	2.00	0.00	1.00	0.32	NS
8 Hours	1.97	0.18	2.00	0.00	1.00	0.32	NS
10 Hours	1.23	0.43	2.00	0.00	9.76	0.0001	NS
12 Hours	1.00	0.00	1.50	0.51	5.39	0.0001	NS
16 Hours	1.00	0.00	1.00	0.00	-	-	



Most of the patients in bupivacaine group had ramsay sedation score ranging from 1.00 to 2.03 between 0 minutes and 16 hours. Similarly in buprenorphine group ramsay sedation score ranging from 1.00 to 2.00 between 0 minutes and 16 hours. The association between the intervention groups and ramsay sedation score is considered to be not statistically significant since $p > 0.05$ as per student t-test.

RESCUE ANALGESIC REQUIREMENT (HOURS)

Group	Mean	Standard Deviation
GROUP-I	7.10	0.40
GROUP-II	13.10	0.96
t-value	31.58	
p-value	0.0001	
Significance	**Significant**	



The mean time at which first rescue analgesic given in bupivcaine group is 7.10 hours while in buprenorphine group, the mean time is 13.10 hours.The association between the intervention groups and the first rescue analgesic ime is considered to be statistically significant since p value is <0.05 as per student t - test.

DISCUSSION

The aim of the study was to compare the ultrasound guided supraclavicular brachial plexus block using bupivacaine alone with combination of bupivacaine and buprenorphine in patients undergoing elective upper limb orthopaedic surgeries with respect to,

1) Postoperative analgesia using visual analogue pain scale.

2) Postoperative analgesic initiation time.

3) Intraoperative hemodynamics.

In this study, ultrasound guided supraclavicular brachial plexus block is used due to its gained popularity because of less failure rate and less complications with this technique.

Krutika et al (2013) performed ultrasound guided supraclavicular brachial plexus block and compared it with nerve stimulator guided technique. Group I – USG guided technique was used and Group –II nerve stimulator technique was used. They compared block execution time, time of onset of sensory and motor blockade, quality of blockade and success rates. From this study they concluded that USG guided technique is quick to perform with improved safety in positioning the needle and accuracy in visualizing the anatomical structures. The success rate in ultrasound group was 96.67% compared to 80% in nerve stimulator group.[16]

Vienna et al (1994), developed the ultrasound guided technique for the supraclavicular approach and demonstrated the high success rate with this technique.

Stein et al (1997) demonstration of presence of peripheral opioid receptors on sensory nerve terminals prompted the use of opioids in peripheral nerve blocks for the prolongation of post operative analgesia.[25]

In the study of Kenneth D.Candido et al(2002), the mean weight of patients was 74.65 kgs and 0.3 mg of buprenorphine was used. In this study, the mean weight of the patients was 58.4 kgs and buprenorphine was used on a weight basis at 3µg/kg.[6]

In this study, the parameters compared are onset of sensory and motor blockade, duration of analgesia, intraoperative and post operative heart rate, systolic blood pressure, diastolic blood pressure, mean blood pressure, Spo2, respiratory rate, Ramsay sedation scale, Visual analogue scale and First rescue analgesic time.

In this study,

ONSET OF SENSORY BLOCKADE:

The mean time of onset of sensory blockade in Group I (bupivacaine alone) is 11.67 minutes. The mean onset time in Group II (bupivacaine and buprenorphine) is 11.50 minutes which is statistically insignificant as pointed out in the study by Bharat et al(2013).[4]

ONSET OF MOTOR BLOCKADE:

The mean time of onset of motor blockade in Group I (bupivacaine alone) patients is 9.27 minutes. The mean time of onset in Group II (bupivacaine and buprenorphine) patients is 9.70 minutes which is statistically significant.But in the study conducted by Bharat et al (2013), there was no significant difference in the onset time of motor blockade between the two groups.[4]

DURATION OF ANALGESIA:

The mean duration of analgesia in Group I (bupivacaine alone) is 5.03 hours. The mean duration of analgesia in Group II(bupivacaine and buprenorphine) is 10.08 hours which is statistically significant. Thus addition of buprenorphine provides a significant prolongation of analgesia as pointed out in the study by Kenneth D.Candido and colleagues (2002).[6] In their study, adddition of buprenorphine 0.3 mg conferred analgesia of 22.3 hours compared to 6.6 hours in local anaesthetic alone. This is also consistent with the study done by Bharat et al(2013).[4] In this study, it was found out that, there was statistically significant difference in the duration of analgesia between the two groups.

PULSE RATE:

Most of the bupivacaine group patients had mean pulse rate ranging from 78.40 bpm to 79.93 bpm whereas buprenorphine group patients had mean pulse rate ranging from 78.83 bpm to 80.63 bpm which is statistically insignificant as pointed out in the study by Bharat et al (2013)[4] and J.E.Bazin et al (1997).[3]

SYSTOLIC BLOOD PRESSURE:

In bupivacaine group, patients had mean systolic blood pressure ranging from 119.13 mmHg to 120.13 mmHg whereas in buprenorphine group patients, the mean blood pressure ranging from 116.93 mmHg to 118.80 mmHg which is statistically insignificant as found out in the study by J.E.Bazin et al (1997).[3]

DIASTOLIC BLOOD PRESSURE:

In bupivacaine group, patients had mean diastolic blood pressure ranging from 73.07 mmHg to 74.20 mmHg whereas, in buprenorphine group patients, the mean diastolic blood pressure was ranging from 73.27 mmHg to 74.0 mmHg which is statistically insignificant as found out in the study by J.E.Bazin et al (1997).[3]

MEAN ARTERIAL BLOOD PRESSURE:

Most of the bupivacaine group patients had mean arterial pressure ranging from 88.03 mmHg to 89.13 mmHg whereas in buprenorphine group, patients had mean arterial pressure ranging from 87.53 mmHg to 89.10 mmHg which is statistically insignificant as pointed out by Bharat and his colleagues (2013).[4]

SPO2:

Most of the bupivacaine group patients had mean spo2 ranging from 98.97% to 99% whereas in buprenorphine group patients, the mean spo2 was 99% which is statistically insignificant as found out by Bharat et al (2013).[4]

RESPIRATORY RATE:

In bupivacaine group, patients had respiratory rate ranging from 15.37 breaths/min to 15.43 breaths/min whereas in buprenorphine group patients, the respiratory rate ranging from 15.33 breaths/min to 15.40 breaths/min which is statistically insignificant as pointed out in the study done by Bharat et al (2013).[4]

VISUAL ANALOGUE SCORE:

In bupivacaine group, the mode for visual analogue score is 6,7,8 hours whereas in buprenorphine group, mode for visual analogue score is 12 hours which is statistically significant.After 12 hours there was no difference in visual analogue score between the two groups. This result is consistent with the study by Bharat et al (2013), where the visual analogue score was lower for buprenorphine group than plain bupivacaine group at all times except at 24 hours at which there was no difference between the two groups.[4]

RAMSAY SEDATION SCORE:

In bupivacaine group, most of the patients had ramsay sedation score ranging from 1.00 to 2.03whereas in buprenorphine group, ramsay sedation score ranging from 1.00 to 2.00 which is statistically insignificant. But, in the study by Bharat et al (2013), there was higher sedation score was achieved in buprenorphine group compared to bupivacaine group.[4]

FIRST RESCUE ANALGESIC TIME:

The mean time at which first rescue analgesic given in bupivcaine group is 7.10 hours with SD 0.4, while in buprenorphine group, the mean time is 13.10 hours witg SD 0.96, which is statistically significant. This is consistent with the study by Bharat et al (2013), where the mean time of initiation of first rescue analgesic is 9 hours with SD 2.7 in bupivacaine group, whereas in buprenorphine group, the mean time was 18 hours with SD 6.3.[4]

SIDE EFFECTS:

None of the patients in the two groups showed side effects like nausea, vomiting, hypotension, constipation, sedation or respiratory depression as pointed out in the study by Kenneth D. Candido et al (2002), where none of the patients developed opioid related side effects.[4]

SUMMARY

For ultrasound guided supraclavicular brachial plexus block, one group received bupivacaine alone anad other group received bupivacaine and buprenorphine. On comparing the two groups, it was found out that,

Onset of sensory blockade was similar in both the groups.

Onset of motor blockade was found to be earlier in bupivacaine alone group.

Duration of analgesia was significantly prolonged in group that received buprenorphine compared to bupivacaine alone.

The need for rescue analgesic is significantly delayed in the group that received buprenorphine compared to bupivacaine alone.

There was no significant difference in the hemodynamic parameters in both the groups.

There was no opioid related side effects in both the groups.

No other complications were seen in both the groups.

CONCLUSION

From this study, it is inferred that the addition of buprenorphine to 0.25% bupivacaine definitely prolonged the duration of analgesia by two fold when compared to 0.25% bupivacaine alone.

To conclude that, addition of 3µ/kg of buprenorphine to 0.25% bupivacaine in ultrasound guided supraclavicular brachial plexus block confers a significant advantage over plain bupivacaine in terms of duration of analgesia and need for rescue analgesia.

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PROFORMA

NAME: AGE/SEX:

IP.NO: WEIGHT:

DIAGNOSIS SURGICAL PROCEDURE DONE:

PRE OP ASSESSMENT:

HISTORY

EXAMINATION:

AIRWAY:

ASA status:

PREOP PR: PREOP BP:

MEASURES OF STUDY OUTCOME:

ONSET OF SENSORY BLOCKADE:

Time in mins	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Loss of pinprick sensation															

ONSET OF MOTOR BLOCKADE:

Time in mins	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Loss of shoulder abduction															

HEMODYNAMIC PARAMETERS:

Time in mins	0	1	5	10	15	30	60	90	120	240
Heart rate										
Systolic BP										
Diastolic BP										
Mean BP										
Respiratory rate										
Spo2										

DURATION OF SURGERY (IN MINS) :

POST OPERATIVE PARAMETERS:

Time in hours	0	1	2	3	4	5	6	8	10	12	16	20	24
Visual analogue score													
Ramsay sedation score													
Side effects													

DUARATION OF ANALGESIA:

POST OP ANALGESIC INITIATION TIME:

INFORMATION TO PARTICIPANTS

Investigator **:** Dr.G.ARCHANA

Name of the Participant:

Title **"A Prospective, randomized study comparing ultrasound guided supraclavicular brachial plexus block using bupivacaine with bupivacaine and buprenorphine in patients undergoing elective upper limb orthopaedic surgeries"**

You are invited to take part in this research study. We have got approval from the IEC. Your are asked to participate because you satisfy the eligibility criteria .We want to compare and study the analgesic efficacy of bupivaccaine with bupivaccaine and buprenorphine in supraclavicular brachial plexus block.

What is the Purpose of the Research:

For upper limb orthopaedic surgeries, patient is given ultrasound guided supraclavicular brachial plexus block either using bupivaccaine or bupivaccaine and buprenorphine. This study is done to compare the supraclavicular block using bupivaccaine with bupivaccaine and buprenorphine with respect to,

1. Intraoperative hemodynamics
2. Post operative analgesia using visual analogue scale pain score.
3. Post op analgesic initiation time

The Study Design: 60 patients presenting for elective upper limb orthopaedic surgeries were randomly assigned to two groups .

Group1- preoperative supraclavicular block using bupivaccaine .

Group 2- preoperative supraclavicular block using bupivaccaine and buprenorphine.

Benefits: Pre operative supraclavicular block , maintains intra operative hemodynamics, causes excellent & prolonged post operative pain releif.

Problems associated with supraclavicular blocks are avoided.

Discomforts and risks :

May cause nausea, vomiting, constipation- prevention given with appropriate drugs.

May cause respiratory depression –patients with pulmonary fibrosis, COPD are excluded from study.

May cause pneumothorax- this will be prevented by using ultrasound guided technique.

This intervention has been shown to be well tolerated as shown by previous studies. And if you do not want to participate you will have alternative of setting the standard treatment and your safety is our prime concern.

Confidentiality of data and details of study and patients details concerned with this research will be strictly maintained.

All tests , medicine,and medical services concerned with this research will be provided free of cost to the patient.

Time :

Date :

Place :

Signature / Thumb Impression of Patient

Patient Name:

Signature of the Investigator : _____

Name of the Investigator : _____

PATIENT CONSENT FORM

Study title **"A Prospective, randomized study comparing ultrasound guided supraclavicular brachial plexus block using bupivacaine with bupivacaine and buprenorphine in patients undergoing elective upper limb orthopaedic surgeries"**

Study center: INSTITUTE OF ANAESTHESIOLOGY AND CRITICAL CARE, MADRAS MEDICAL COLLEGE & GOVT GENERAL HOSPITAL, CHENNAI 600003.

Participant name : Age: Sex:

I.P.No:

I confirm that I have understood the purpose of procedure for the above study . I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the pitfall in the procedure. I have been explained about the safety,advantage and disadvantage of the technique.

I understand that my participation in the study is voluntary and that I am free to withdraw at anytime without giving any reason.

I understand that investigator ,regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in

relation to it, even if I withdraw from the study . I understand that my identity will not be revealed in any information released to third parties or published , unless as required under the law . I agree not to restrict the use of any data or results that arise from the study .

Time:

Date: Signature /

thumb impression of patient

Place: Patient

name:

Signature of the investigator:

Name of the investigator:

ஆராய்ச்சி தகவல் தாள்

ஆராய்ச்சி தலைப்பு

கைகளின் எலும்பில் மேற்கொள்ளும் அறுவை சிகிச்சையின்போது வலி இல்லாமல் இருப்பதற்கு மயக்க மருந்து கழுத்தில் சுப்ராகிளாவிகுளார் ப்ளாக் மூலம் புபிவெகெயின் அல்லது புபிவெகெயின் மற்றும் புப்ரினாற்ஃபின் மருந்துக் கலவை கொடுப்பதை ஒப்பிடுதல்.

ஆராய்ச்சியாளர் பெயர் : மருத்துவர்.கோ.அர்ச்சனா

பங்கேற்பாளர் பெயர் :

ஆராய்ச்சியின் நோக்கம்

இவ்வாராய்ச்சியின் மூலமாக நான் ஆய்வு செய்ய இருப்பவை,

1) அறுவை சிகிச்சையின்போது இரத்த அழுத்தம் மற்றும நாடித்துடிப்பின் மாற்றங்கள்.

2) அறுவை சிகிச்சைக்குப் பிந்தைய வலி நிவாரண அளவு (விசுவல் அனலாக் அளவுகோல் மூலமாக).

3) அறுவை சிகிச்சைக்குப்பின் முதன்முறை வலி நிவாரணி தேவைப்படும் நேரம்.

ஆய்வு முறை

ஆய்வில் பங்குபெறும் நோயாளிகள் இரண்டு குழுக்களாகப் பிரிக்கப்படுவர்.

குழு-1 அறுவை சிகிச்சைக்கு முன் கழுத்தில் சுப்ராகிளாவிகுளார் பிளாக் மூலம் புபிவெகெயின் மருந்து கொடுக்கப்படும்

குழு-2 அறுவை சிகிச்சைக்கு முன் கழுத்தில் சுப்ராகிளாவிகுளார் பிளாக் மூலம் புபிவெகெயின் மற்றும் புப்ரினாற்ஃபின் மருந்துக் கலவை கொடுக்கப்படும்.

நன்மைகள்

அறுவை சிகிச்சையின்போது இதயத்துடிப்பு மற்றும் இரத்த அழுத்தம் ஆகியவற்றை சீராக வைக்க உதவும்.

அறுவை சிகிச்சைக்குப்பிறகு நீண்ட நேரம் வலி நிவாரணம் அளிக்கக் கூடியது.

முழு மயக்கம் கொடுக்க வேண்டியதை தவிர்த்து அதனால் ஏற்படும் பக்கவிளைவுகளும் தவிர்க்கப்படும்.

பக்கவிளைவுகள்

தலைவலி மற்றும் வாந்தி வருவது போன்ற உணர்வு ஏற்படுத்தலாம். இதை தடுக்க அறுவை சிகிச்சையின் போது மருந்து வழங்கப்படும்.

சுவாச எண்ணிக்கை குறையலாம். இது தீவிரமாக கண்காணிப்பட்டு தக்க வைத்தியம் அளிக்கப்படும். சுவாசப் பிரச்சனை உள்ளவர்கள் இவ்வாய்விலிருந்து விலக்கப்படுவார்கள்.

மலச்சிக்கல் ஏற்படலாம். இதற்கு அறுவை சிகிச்சைக்குப்பின் மருந்து வழங்கப்படும்.

தூக்கமின்மை ஏற்படலாம். இது அரிய பக்கவிளைவாகும்.

இந்த ஆய்வு சம்பந்தமான எல்லா புள்ளி விவரங்கள் மற்றும் நோயாளிகளின் விவரங்கள் ரகசியமாக வைக்கப்படும். இந்த ஆய்வு சம்பந்தப்பட்ட எல்லா பரிசோதனைகள், மருந்துகள் மற்றும் மருத்துவ சேவைகள் அனைத்தும் நோயாளிகளுக்கு இலவசமாக வழங்கப்படும்.

ஆய்வாளரின் பெயர் **பங்குபெறுபவரின் பெயர்**

ஆய்வாளரின் கையொப்பம் **பங்குபெறுபவரின் கையொப்பம்**

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராயச்சியின் தலைப்பு

கைகளின் எலும்பில் மேற்கொள்ளும் அறுவை சிகிச்சையின்போது வலி இல்லாமல் இருப்பதற்கு மயக்க மருந்து கழுத்தில் சுப்ராகிளாவிகுளார் ப்ளாக் மூலம் புபிவெகெயின் அல்லது புபிவெகெயின் மற்றும் புப்ரினாற்ஃபின் மருந்துக் கலவை கொடுப்பதை ஒப்பிடுதல்.

ஆய்வு நிலையம் : மயக்கவியல் துறை, சென்னை மருத்துவக் கல்லூரி சென்னை - 3.

பங்கு பெறுவரின் பெயர் :

பங்குபெறுபவரின் எண் :

பங்குபெறுபவர் இதனை (✓) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கின்றேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் 'இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிகிறேன். ☐

அறுவை சிகிச்சையின்போது மயக்கமருந்து கழுத்தில் சுப்ராகிளாவிகுளார் பிளாக் மூலம் புபிவெகெயின் அல்லது புபிவெகெயின் மற்றும் புப்ரினாற்ஃபின் மருந்துக் கலவை கொடுக்கப்படும் என்பதை அறிந்துகொண்டேன். இதனால் உடலுக்கு எந்தவிதமான உபாதைகளும் இருக்காது என்பதை அறிந்துகொண்டு இந்த ஆய்வில் பங்குபெற முழு மனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின் கையொப்பம் ..... இடம்..... தேதி.....

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம்..... தேதி.....

ஆய்வாளரின் பெயர் .....

284.4

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.Archana.G.
PG in MD (Anaesthesiology)
Madras Medical College
Chennai 600 003

Dear Dr.Archana.G.

The Institutional Ethics Committee has considered your request and approved your study titled **" A PROSPECTIVE, RANDOMIZED STUDY COMPARING ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK USING BUPIVACAINE WITH BUPIVACAINE AND BUPRENORPHINE IN PATIENT UNDERGOING ELECTIVE UPPER LIMB SURGERIES " NO.02022015**

The following members of Ethics Committee were present in the meeting held on 03.02.2015 conducted at Madras Medical College, Chennai 3.

1. Dr.C.Rajendran, MD	:Chairperson
2. Dr.R.Vimala,MD.,Dean,MMC,Ch-3	: Deputy Chairperson
3. Dr.B.Kalaiselvi,MD.,Vice Principal,MMC,Ch-3	: Member Secretary
4. Dr..R.Nandhini,MD.,Inst.of Pharmacology,MMC	: Member
5. Dr..P.Ragumani, MS., Professor, Inst.of Surgery,MMC	: Member
6. Dr..K.Ramadevi, Director , Inst.of Bio-Chem.MMC	: Member
7. Dr..Saraswathy,MD.,Director,Pathology, MMC	: Member
8. Dr.Md.Ali, MD., DM.,Prof.&HOD of Medl.GE,MD.MMC	: Member
9. Dr.S.G.Sivachidambaram,Director I/c, Inst.of Internal Medicine	: Member
10. Thiru S.Rameshkumar	: Lay Person
11.Thiru S.Govindasamy, BA., BL.,	: Lawyer
12.Tmt.Arnold Saulina, MA., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee
MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

Svs 2

												PULSE RATE								SYSTOLIC BLOOD PRESSURE							
S.NO:	Group	Age	Sex	Weight	BMI	ASA status	Diagnosis	Procedure	onset of sensory blockade (in mins)	onset of motor blockade (in mins)	Duration of analgesia (in hrs)	PR-BL	PR-5	PR-10	PR-15	PR-30	PR-60	PR-90	PR-120	SBP-BL	SBP-5	SBP-10	SBP-15	SBP-30	SBP-60	SBP-90	SBP-120
1	I	32	M	55	19.6	I	Rt Distal humerus fracture	Elbow external fixation	11	9	5	68	65	69	65	61	68	64	68	128	130	124	122	120	112	118	120
2	I	30	F	60	18.06	II	Lt shaft of humerus fracture	ORIF with plating	10	8	5.1	78	72	74	68	70	74	72	71	122	122	120	120	116	114	120	120
3	I	36	F	60	20.02	I	Rt distal radius fracture	K wire fixation	12	9	5.2	92	90	91	88	88	86	90	90	124	122	116	120	124	126	120	116
4	I	19	M	58	19	I	Rt olecranon fracture	Modified tension band wiring	13	11	5.3	110	110	108	108	102	103	102	104	124	124	118	118	112	124	124	120
5	I	56	M	56	19	I	Rt both bone forearm fracture	ORIF with plating	11	9	5	64	62	60	65	66	68	62	64	128	130	128	134	136	126	126	126
6	I	30	F	62	19	II	Lt proximal ulna fracture	ORIF with plating	11	10	5.3	75	76	74	78	80	80	72	70	110	112	116	118	114	112	114	112
7	I	25	F	50	19	II	Lt shaft of humerus fracture	DCP	11	9	5.1	76	72	74	78	76	70	74	72	112	112	112	116	122	120	118	118
8	I	55	F	55	19	I	Rt both bone forearm fracture	ORIF with plating	12	10	5.15	85	84	82	87	80	84	81	85	132	134	124	126	128	132	118	130
9	I	38	M	65	19	I	Rt galeazzi fracture	ORIF with plating	11	9	5.05	78	79	76	80	70	74	73	72	126	124	126	122	120	124	118	118
10	I	20	M	56	19	I	Lt capitulum fracture	Herbert screw fixation	13	9	5.1	65	68	69	67	66	68	74	64	116	114	110	110	106	110	118	114
11	I	33	M	58	19	I	Lt both bone forearm fracture	ORIF with plating	12	10	5.3	78	80	84	85	73	72	80	78	124	126	124	122	116	118	118	112
12	I	21	M	55	19	I	Lt lateral condyle humerus	Recon plating	11	8	4.55	76	76	78	80	83	75	76	77	110	108	114	114	116	112	118	110
13	I	30	M	60	19	II	Lt non union radius	ORIF with plating	12	10	5.01	66	65	70	72	72	71	68	66	122	122	128	124	118	126	118	124
14	I	36	M	62	19	II	Rt distal radius fracture	ORIF with plating	12	9	4.5	97	96	98	95	94	90	89	96	134	136	140	136	134	130	118	132
15	I	45	F	52	19	I	Rt both bone forearm fracture	ORIF with plating	12	10	5.1	76	80	83	82	78	78	77	73	128	126	122	124	126	124	118	120
16	I	48	M	64	19	II	Lt neglected elbow dislocation	ORIF with plating and triceps plasty	11	9	5.1	62	67	68	71	67	65	64	72	114	110	114	114	116	110	118	114
17	I	60	F	54	19	II	Lt non union radius	ORIF with plating	12	10	5.05	102	100	98	100	105	106	97	96	122	124	126	124	122	128	118	118
18	I	21	F	50	19	I	Rt distal radius fracture	ORIF with plating	11	9	5.15	86	87	83	82	78	76	81	80	122	120	118	118	118	114	118	122
19	I	20	F	54	19	I	Rt both bone forearm fracture	ORIF with plating	12	10	4.5	76	70	74	72	68	69	66	75	114	112	116	114	120	112	118	122
20	I	27	M	56	19	I	Rt shaft of humerus fracture	ORIF with plating	11	8	5.3	74	68	69	70	72	77	72	74	124	122	118	116	124	122	118	124
21	I	32	M	65	19	I	Lt distal radius fracture	K wire fixation	12	10	5.25	66	64	61	70	74	68	70	72	112	110	106	112	110	114	118	114
22	I	37	M	56	19	I	Rt both bone forearm fracture	ORIF with plating	11	9	5.1	74	72	73	68	66	72	77	70	112	110	114	122	126	118	118	120
23	I	35	M	55	19	I	Rt distal humerus fracture	ORIF with plating	13	10	4.55	88	87	79	80	82	81	84	88	112	110	116	118	122	118	118	120
24	I	23	M	52	19	I	Rt both bone forearm fracture	ORIF with plating	12	9	5.15	71	72	77	73	80	83	82	80	122	120	118	116	112	116	118	124
25	I	20	M	56	19	I	Lt shaft of humerus fracture	ORIF with plating	11	9	5.2	92	90	89	87	93	92	95	90	114	112	114	116	116	118	118	116
26	I	30	M	58	19	I	Lt olecranon fracture	Modified tension band wiring	12	9	5	72	71	76	80	82	83	78	76	122	122	120	126	128	120	118	118
27	I	33	M	54	19	I	Rt shaft of humerus fracture	ORIF with plating	13	10	4.55	89	87	76	65	64	65	64	76	110	112	114	114	112	128	118	116
28	I	48	M	60	19	II	Lt both bone forearm fracture	ORIF with plating	12	9	5	78	76	75	80	82	84	91	82	120	120	122	118	116	114	118	122
29	I	30	F	55	19	I	Rt both bone forearm fracture	ORIF with plating	11	8	5.1	98	97	88	87	91	90	92	87	122	128	130	126	124	122	118	124
30	I	30	F	54	19	I	Lt distal radius fracture	ORIF with plating	12	9	5	86	86	85	78	77	83	86	84	122	112	114	110	110	116	118	108

DIASTOLIC BLOOD PRESSURE								MEAN ARTERIAL PRESSURE								SPo2													RESPIRAT						
DBP-BL	DBP-5	DBP-10	DBP-15	DBP-30	DBP-60	DBP-90	DBP-120	MAP-BL	MAP-5	MAP-10	MAP-15	MAP-30	MAP-60	MAP-90	MAP-120	S-BL	S-5	S-10	S-15	S-30	S-60	S-90	S-120	S-4hrs	S-6	S-8	S-10	S-12	S-16	RR-BL	RR-5	RR-10	RR-15	RR-30	RR-60	RR-90
74	80	72	72	72	70	72	70	92	96	89	88	88	84	87	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	15	15	15	15	15
80	72	74	76	70	72	72	70	94	88	89	90	85	86	88	86	99	99	99	99	98	99	99	98	99	99	99	99	98	99	15	15	15	16	16	16	15
80	82	88	86	84	82	80	80	94	95	97	97	97	96	93	92	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	15	15	16	16
80	82	80	78	76	76	78	80	94	96	92	91	88	92	93	93	99	99	99	98	99	99	99	99	99	99	99	99	99	99	15	15	15	15	15	15	15
78	76	78	74	72	78	70	74	94	94	94	94	93	94	88	91	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	16	16	15	15	15	15
76	72	74	70	70	72	70	70	87	85	88	86	84	85	84	84	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	16	15	16	15	16	15
76	78	74	76	72	70	72	70	88	89	86	89	88	86	87	86	99	99	99	98	99	99	99	99	99	99	99	99	99	99	15	16	16	15	15	15	15
80	82	80	86	82	84	80	82	97	99	94	99	97	100	96	98	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	15	16	16	15	15
78	74	78	72	70	70	72	74	94	90	94	88	86	88	88	88	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	16	15	15	16	15	16
74	72	72	76	74	72	74	74	88	86	84	87	84	84	86	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	16	16	15	16	15
80	78	76	70	74	72	72	74	94	94	92	87	88	87	86	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	15	15	16	15	15
72	68	66	70	66	68	64	66	84	81	82	84	82	82	80	80	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	16	16	15	16	16
72	74	70	72	72	74	74	72	88	90	89	89	87	91	90	89	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	15	15	16	15
80	82	84	82	80	82	84	84	98	100	102	100	98	98	100	100	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	15	16	15	15	15
74	70	74	68	72	70	72	70	92	88	90	86	90	88	88	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	16	15	15	16	16
70	68	62	60	62	64	66	68	84	82	79	78	80	79	81	83	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	16	16	16	15	16
78	76	78	72	70	72	74	72	92	92	94	89	87	90	90	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	15	16	15	16
68	68	70	64	68	68	70	72	86	85	86	82	84	83	86	88	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	16	15	15	16	16
70	68	64	66	70	68	66	68	84	82	81	82	86	82	83	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	15	16	15	16
74	74	74	70	72	76	76	78	90	90	88	85	89	91	90	93	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	15	16	16	15	15
68	68	70	66	68	70	72	74	82	82	82	81	82	84	86	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	15	15	15	15	16
68	70	72	70	74	74	72	70	82	83	86	87	91	88	86	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	15	15	15	15	16
72	70	74	76	76	72	74	72	85	83	88	90	91	87	88	88	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	16	15	15	15	15
76	74	72	74	76	72	74	72	91	89	87	88	88	86	88	89	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	16	16	16	15
68	66	64	70	70	72	72	72	83	81	80	85	85	87	86	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	16	15	16	15	15
70	72	76	74	72	70	72	70	87	88	90	91	90	86	87	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	15	16	15	16	16
66	68	66	70	76	74	74	72	80	82	82	84	88	92	88	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	15	15	16	15
68	70	74	76	78	74	76	72	85	86	90	90	90	87	90	88	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	15	15	15	15	15
80	82	78	84	82	80	78	80	94	97	95	98	96	94	92	94	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	16	15	15	15	16
76	74	78	80	72	74	72	74	91	86	90	90	84	88	86	85	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	16	16	16	16	15

TORY RATE							VISUAL ANALOGUE SCALE										RAMSAY SEDATION SCALE												
RR-120	RR-4hrs	RR-6	RR-8	RR-10	RR-12	RR-16	VAS-2	VAS-4	VAS-5	VAS-6	VAS-7	VAS-8	VAS-10	VAS-11	VAS-12	VAS-16	R-0	R-15	R-30	R-1	R-2	R-4	R-6	R-8	R-10	R-12	R-16	COMPLICATIONS	RESCUE ANALGESIC REQUIREMENT(HR)
15	15	16	15	15	15	15	0	0	2	4							1	2	2	2	2	2	2	1	1	1	1	NIL	6
15	16	15	16	15	15	17	0	0	1	2	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
15	16	16	15	15	16	15	0	0	0	2	4						1	1	2	2	2	2	2	2	1	1	1	NIL	7
15	15	16	15	16	16	15	0	0	0	2	4						1	2	2	2	2	3	2	2	2	1	1	NIL	7
15	15	15	16	15	16	15	0	0	1	3	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
16	16	15	15	15	15	16	0	0	0	2	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
15	15	15	16	16	16	16	0	0	1	2	4						1	2	2	2	2	2	2	2	2	1	1	NIL	7
16	16	16	15	16	15	16	0	0	0	2	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
16	16	15	15	16	15	15	0	0	1	3	4						1	2	2	2	2	2	2	2	2	1	1	NIL	7
15	15	15	15	15	16	15	0	0	1	2	4						1	1	2	2	2	2	2	2	1	1	1	NIL	7
15	16	15	16	15	15	15	0	0	0	2	3	4					1	2	2	2	2	2	2	2	1	1	1	NIL	8
15	15	15	15	15	15	15	0	0	1	3	4						1	1	2	2	2	2	2	2	1	1	1	NIL	7
16	16	16	16	16	16	16	0	0	2	3	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
16	15	15	16	15	16	15	0	0	2	3	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
15	16	16	15	16	16	16	0	0	1	2	2	4					1	2	2	2	2	2	2	2	1	1	1	NIL	8
15	15	16	16	16	16	16	0	0	1	2	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
16	15	15	16	15	15	15	0	0	1	2	4						1	1	2	2	2	2	2	2	2	1	1	NIL	7
16	16	15	15	15	17	15	0	0	1	2	3	4					1	2	2	2	2	2	2	2	1	1	1	NIL	8
15	15	15	15	15	15	15	0	0	2	3	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
15	15	15	16	15	15	15	0	0	0	2	3	4					1	2	2	2	2	2	3	2	1	1	1	NIL	8
16	16	15	15	15	16	15	0	0	0	2	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
15	15	15	15	15	15	15	0	0	1	3	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
15	15	16	15	16	15	16	0	0	1	2	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
16	15	15	15	15	15	16	0	0	0	2	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
15	15	16	15	16	15	15	0	0	0	2	4						1	2	2	2	2	2	2	2	2	1	1	NIL	7
15	15	15	15	15	15	15	0	0	2	3	4						1	2	2	2	2	2	2	2	2	1	1	NIL	7
15	15	15	15	15	15	16	0	0	1	2	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
16	16	15	16	15	14	15	0	0	1	3	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
16	15	16	16	16	15	16	0	0	1	3	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7
15	15	16	15	16	15	16	0	0	1	2	4						1	2	2	2	2	2	2	2	1	1	1	NIL	7

S.NO:	Group	Age	Sex	Weight	BMI	ASA status	Diagnosis	Procedure	onset of sensory blockade (in mins)	onset of motor blockade (in mins)	Duration of analgesia (in hrs)	PR-BL	PR-5	PR-10	PR-15	PR-30	PR-60	PR-90	PR-120	SBP-BL	SBP-5	SBP-10	SBP-15	SBP-30	SBP-60	SBP-90	SBP-120
1	II	29	M	65	21	I	Lt both bone forearm fracture	ORIF with plating	11	10	9.45	106	100	98	102	97	96	103	102	114	112	118	120	122	116	110	116
2	II	30	F	62	20.5	I	Lt proximal ulna fracture	ORIF with plating	12	11	10.1	72	78	76	80	82	76	77	78	124	122	122	112	114	116	118	120
3	II	45	M	64	20	II	Lt shaft of humerus fracture	DCP	10	9	10.3	88	90	92	86	93	94	86	85	128	130	126	122	126	126	128	128
4	II	29	M	58	19.6	I	Rt both bone forearm fracture	ORIF with plating	11	9	11	67	68	70	73	72	66	65	71	118	114	110	114	118	112	110	114
5	II	31	F	55	18.8	I	Rt Distal humerus fracture	Elbow external fixation	12	10	10	77	76	78	80	72	73	71	70	112	110	114	120	112	112	108	110
6	II	37	M	65	21.4	I	Lt shaft of humerus fracture	ORIF with plating	10	9	10.15	67	66	62	68	70	65	64	62	130	130	124	120	118	118	116	114
7	II	24	F	54	20	I	Rt distal radius fracture	K wire fixation	11	9	9.5	78	76	77	80	83	82	75	83	124	124	126	118	116	114	112	114
8	II	28	M	65	19.2	I	Rt olecranon fracture	Modified tension band wiring	12	10	10.3	88	86	90	83	84	92	85	88	114	114	110	112	114	112	110	114
9	II	55	F	58	19.5	II	Lt non union radius	ORIF with plating	13	10	10.4	72	77	76	73	71	68	69	70	124	126	130	132	128	128	130	128
10	II	55	M	60	20	I	Rt distal radius fracture	ORIF with plating	12	10	10.05	89	90	87	92	94	87	92	95	116	114	112	116	120	122	118	120
11	II	45	F	55	18.6	I	Rt both bone forearm fracture	ORIF with plating	11	10	9.55	70	71	76	68	76	75	72	74	112	110	112	116	114	118	124	122
12	II	57	M	64	20.6	I	Lt non union radius	ORIF with plating	11	9	9.45	96	95	90	94	89	93	91	90	116	118	116	112	122	118	112	116
13	II	21	M	56	20	I	Rt distal radius fracture	ORIF with plating	12	10	10	102	100	99	97	98	95	96	99	114	110	106	110	112	116	116	116
14	II	52	M	64	21	II	Rt both bone forearm fracture	ORIF with plating	11	9	10.2	78	80	76	81	80	82	76	74	128	126	122	118	120	118	116	116
15	II	35	F	55	18.6	I	Rt both bone forearm fracture	ORIF with plating	12	10	10.15	92	89	91	88	90	92	93	88	110	108	108	112	114	114	112	114
16	II	40	M	62	20.05	I	Rt shaft of humerus fracture	ORIF with plating	10	9	10	86	81	78	80	82	84	88	80	124	126	118	118	112	118	120	118
17	II	29	M	56	19	I	Lt distal radius fracture	K wire fixation	10	8	9.5	76	71	70	72	73	71	76	80	124	122	118	116	112	118	120	120
18	II	20	M	58	19.6	I	Rt both bone forearm fracture	ORIF with plating	12	9	10.3	81	80	78	76	77	69	70	72	110	108	110	114	116	114	112	114
19	II	32	M	65	20.8	I	Lt olecranon fracture	Modified tension band wiring	14	12	10.4	90	91	98	95	89	92	90	94	126	124	128	118	116	118	126	122
20	II	58	M	60	19.4	II	Lt distal radius fracture	K wire fixation	12	10	10.5	86	85	84	78	80	82	76	77	114	112	110	122	118	114	112	114
21	II	40	F	52	18.9	I	Rt both bone forearm fracture	ORIF with plating	12	10	9.5	71	70	72	71	74	76	70	68	126	128	132	126	124	122	116	118
22	II	25	M	55	18.05	I	Rt distal humerus fracture	ORIF with plating	11	9	11.1	86	84	86	80	78	76	82	84	114	110	108	114	112	116	118	120
23	II	35	F	58	19.4	I	Rt both bone forearm fracture	ORIF with plating	11	10	10.4	62	65	67	60	69	72	71	70	120	122	118	112	122	118	116	110
24	II	38	M	62	20.4	I	Lt shaft of humerus fracture	ORIF with plating	12	11	10.45	76	78	82	80	77	73	71	76	118	112	112	120	122	116	110	108
25	II	42	M	55	19	I	Lt non union radius	ORIF with plating	11	10	10.3	80	82	81	78	79	76	80	84	118	124	118	114	110	114	118	116
26	II	45	M	65	21	II	Rt proximal ulna fracture	ORIF with plating	12	9	10.15	72	71	68	74	76	75	78	80	120	120	118	116	120	120	122	114
27	II	55	M	58	20.02	II	Rt Distal humerus fracture	ORIF with plating	12	10	9.5	76	77	80	81	76	73	72	76	112	110	112	108	114	118	120	118
28	II	31	M	54	18.4	I	Lt shaft of humerus fracture	DCP	11	9	9.45	66	65	70	65	64	71	72	70	114	116	112	110	114	114	116	116
29	II	37	M	55	18.5	I	Rt both bone forearm fracture	ORIF with plating	12	10	10	92	90	89	88	84	85	82	81	112	110	114	114	108	114	122	116
30	II	45	M	60	20	I	Rt distal radius fracture	K wire fixation	12	10	10.1	76	75	78	73	80	74	72	71	128	126	128	130	126	124	120	122

DBP-BL	DBP-5	DBP-10	DBP-15	DBP-30	DBP-60	DBP-90	DBP-120	MAP-BL	MAP-5	MAP-10	MAP-15	MAP-30	MAP-60	MAP-90	MAP-120	S-BL	S-5	S-10	S-15	S-30	S-60	S-90	S-120	S-4hrs	S-6	S-8	S-10	S-12	S-16	RR-BL	RR-5	RR-10	RR-15	RR-30	RR-60	RR-90
72	72	74	72	72	74	72	70	86	85	88	88	88	88	84	85	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	16	15	15	16	15
76	74	76	74	74	72	74	72	92	90	91	86	87	86	88	88	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	16	16	16	15	16
78	76	78	76	74	74	72	74	94	94	94	91	91	91	90	92	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	15	16	15	16	16
70	72	72	74	76	72	74	74	86	86	84	87	90	85	86	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	15	16	15	15
74	76	74	76	78	74	74	72	86	87	87	90	89	86	85	84	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	15	15	16	15	15
76	78	76	78	76	74	74	72	94	95	92	92	90	88	88	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	16	15	15	15	15	16
76	76	74	74	76	76	76	74	92	92	91	88	89	88	88	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	16	15	15	16	16
74	76	74	72	74	74	72	72	87	88	86	85	87	86	84	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	16	16	16	15	15
78	76	74	74	76	74	74	72	93	92	92	93	93	92	92	90	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	15	15	15	15	16
86	86	82	80	78	74	76	78	96	95	92	92	92	90	90	92	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	15	16	16	15
74	72	76	72	74	72	74	72	86	84	88	86	87	87	90	88	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	16	16	15	15	16
76	74	74	74	74	74	74	72	89	88	88	86	90	88	86	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	15	15	16	15	15
66	68	68	70	74	72	72	70	82	82	84	86	86	86	86	85	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	15	15	16	16
76	78	74	74	74	76	72	74	93	94	90	88	89	90	86	88	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	16	15	15	15	15
74	72	72	74	72	74	72	74	86	84	84	86	86	87	85	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	16	15	15	15	16
74	72	74	76	76	74	74	72	90	90	88	90	88	88	89	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	16	15	15	15
76	72	74	74	76	74	72	72	92	88	88	88	88	88	88	88	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	15	15	16	16	15
72	66	70	74	76	74	72	74	84	80	83	87	89	87	85	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	15	16	15	15	16
76	74	72	74	76	72	74	74	92	90	90	88	89	87	91	90	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	16	15	15	15
72	70	74	76	72	72	74	74	86	84	86	91	87	86	86	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	15	15	15	16	15
74	74	76	76	72	74	72	72	91	92	94	92	89	90	86	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	15	16	15	16	16
72	74	70	76	72	74	74	72	86	86	82	88	85	88	88	88	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	15	16	15	16	15	15
76	78	80	76	74	72	74	72	90	92	92	88	90	87	88	84	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	16	16	15	15	16	15
76	74	78	80	74	72	74	76	90	86	89	93	90	86	86	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	16	16	15	16	15	15	16
74	76	76	72	74	72	76	78	88	92	88	86	86	86	90	90	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	16	15	16	16	15
76	72	72	70	72	74	72	72	90	88	87	85	88	89	88	86	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	15	16	16	15	15
74	74	70	72	70	74	72	72	86	86	88	84	84	88	88	87	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	16	15	15	15	15
72	70	66	66	68	70	76	74	86	85	84	86	83	84	89	88	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	15	16	15	16	15
76	78	74	70	70	72	76	76	88	90	87	84	82	86	91	89	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	15	15	16	16	15
74	78	76	72	70	72	74	76	92	94	93	91	88	89	89	91	99	99	99	99	99	99	99	99	99	99	99	99	99	99	15	15	16	15	16	15	15

RR-120	RR-4hrs	RR-6	RR-8	RR-10	RR-12	RR-16	VAS-2	VAS-4	VAS-5	VAS-6	VAS-7	VAS-8	VAS-10	VAS-11	VAS-12	VAS-16	R-0	R-15	R-30	R-1	R-2	R-4	R-6	R-8	R-10	R-12	R-16	COMPLICATIONS	RESCUE ANALGESIC REQUIREMENT(HR)
16	15	15	15	15	16	15	0	0	0	0	0	0	2	3	4		1	2	2	2	2	2	2	2	2	2	1	NIL	12
15	15	16	15	16	15	16	0	0	0	0	0	0	1	2	4		1	2	2	2	2	2	2	2	2	1	1	NIL	12
16	16	15	16	16	16	16	0	0	0	0	0	0	0	1	3		1	2	2	2	2	2	2	2	2	2	1	NIL	13
16	15	16	16	16	16	16	0	0	0	0	0	0	0	1	3		1	2	2	2	2	2	2	2	2	2	1	NIL	13
16	16	15	16	15	15	15	0	0	0	0	0	0	1	3	4		1	2	2	2	2	2	2	2	2	2	1	NIL	12
15	15	15	15	16	15	15	0	0	0	0	0	0	1	2	4		1	2	2	2	2	2	2	2	2	1	1	NIL	12
15	15	15	15	15	16	16	0	0	0	0	0	0	2	3	4		1	2	2	2	2	2	2	2	2	2	1	NIL	12
15	16	16	15	15	15	15	0	0	0	0	0	0	0	1	2		1	2	2	2	2	2	2	2	2	2	1	NIL	15
15	15	15	15	15	15	15	0	0	0	0	0	0	0	2	3		1	2	2	2	2	2	2	2	2	1	1	NIL	14
15	15	16	16	15	15	15	0	0	0	0	0	0	1	3	4		1	2	2	2	2	2	2	2	2	2	1	NIL	12
15	16	16	15	15	16	16	0	0	0	0	0	0	1	2	4		1	2	2	2	2	2	2	2	2	1	1	NIL	13
15	15	15	15	16	15	15	0	0	0	0	0	0	1	2	4		1	2	2	2	2	2	2	2	2	1	1	NIL	13
15	15	16	15	15	16	15	0	0	0	0	0	0	1	2	3		1	2	2	2	2	2	2	2	2	1	1	NIL	14
15	15	16	16	15	15	15	0	0	0	0	0	0	0	2	3		1	2	2	2	2	2	2	2	2	2	1	NIL	15
16	16	15	15	15	16	16	0	0	0	0	0	0	0	1	2		1	2	2	2	2	2	2	2	2	1	1	NIL	14
16	15	16	15	15	16	15	0	0	0	0	0	0	1	2	3		1	2	2	2	2	2	2	2	2	2	1	NIL	12
15	15	15	15	15	15	16	0	0	0	0	0	0	2	3	4		1	2	2	2	2	2	2	2	2	1	1	NIL	13
16	15	15	16	16	15	15	0	0	0	0	0	0	0	1	3		1	2	2	2	2	2	2	2	2	1	1	NIL	13
15	16	15	15	15	15	16	0	0	0	0	0	0	0	1	3		1	1	2	2	2	2	2	2	2	1	1	NIL	14
15	16	15	15	16	15	15	0	0	0	0	0	0	0	1	2		1	2	2	2	2	2	2	2	2	2	1	NIL	14
15	15	15	16	15	16	15	0	0	0	0	0	0	1	2	3		1	2	2	2	2	2	2	2	2	1	1	NIL	14
15	16	15	16	15	15	15	0	0	0	0	0	0	0	1	2		1	2	2	2	2	2	2	2	2	1	1	NIL	14
16	15	16	16	16	15	16	0	0	0	0	0	0	0	1	2		1	1	2	2	2	2	2	2	2	2	1	NIL	14
15	16	16	15	15	15	15	0	0	0	0	0	0	0	1	2		1	2	2	2	2	2	2	2	2	2	1	NIL	14
16	15	15	15	16	15	15	0	0	0	0	0	0	0	1	2		1	2	2	2	2	2	2	2	2	1	1	NIL	13
15	16	15	15	15	16	15	0	0	0	0	0	0	1	2	3		1	2	2	2	2	2	2	2	2	1	1	NIL	12
15	15	16	16	15	15	15	0	0	0	0	0	0	1	2	4		1	2	2	2	2	2	2	2	2	2	1	NIL	12
16	15	15	16	16	15	16	0	0	0	0	0	0	1	2	4		1	2	2	2	2	2	2	2	2	2	1	NIL	13
15	16	16	15	15	16	15	0	0	0	0	0	0	1	2	3		1	2	2	2	2	2	2	2	2	1	1	NIL	13
16	15	15	15	16	15	15	0	0	0	0	0	0	1	2	3		1	1	2	2	2	2	2	2	2	2	1	NIL	12